The Right Ventricle in Cardiac Surgery, a Perioperative Perspective: I. Anatomy, Physiology, and Assessment

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The importance of right ventricular (RV) function in cardiovascular disease and cardiac surgery has been recognized for several years. RV dysfunction has been shown to be a significant prognostic factor in heart failure, congenital heart disease, valvular disease, and cardiac surgery. In the first of our two articles, we will review key features of RV anatomy, physiology, and assessment. In the first article, the main discussion will be centered on the echographic assessment of RV structure and function. In the second review article, pathophysiology, clinical importance, and management of RV failure in cardiac surgery will be discussed.
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The importance of right ventricular (RV) function in cardiac surgery has been recognized for several years. Many studies have, in fact, demonstrated its prognostic value after valvular heart surgery, coronary artery bypass surgery, heart transplantation, and left ventricular (LV) assist device insertion.[1,2] Severe refractory RV failure requiring prolonged inotropic support or RV assist device insertion occurs in approximately 0.1% of patients after cardiotomy, in 2%–3% of patients after heart transplantation, and in 20%–30% of patients receiving a LV assist device.[2] The survival rate associated with severe RV failure may be as low as 25%–30%.[2] This highlights the importance of early diagnosis as well as better preventive and management strategies. In the perioperative and intensive care setting, echocardiography is becoming the mainstay in the assessment of RV function.

We have recently reviewed the role of the RV in cardiovascular disease.[3,4] In this article, we will review RV function from the perspective of the surgeon and anesthesiologist caring for the perioperative patient undergoing cardiac surgery. We will discuss perioperative assessment of RV function, whereas the companion review focuses on the clinical importance of RV dysfunction and on perioperative management.

This article has supplementary material on the Web site: www.anesthesia-analgesia.org.

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RV ANATOMY

The anatomy of the RV is both unique and complex. The RV appears triangular when viewed laterally, whereas in cross-section, it appears crescent shaped.[5] Although the RV appears smaller than the LV in the four-chamber view, RV volume is, in fact, larger than the LV volume. Based on magnetic resonance imaging, the normal range of RV end-diastolic volume (RVEDV) is 49–101 mL/m^2 (males, 55–105 mL/m^2; females, 48–87 mL/m^2), whereas the normal range of LV end-diastolic volume is 44–89 mL/m^2 (males, 47–92 mL/m^2; females, 41–81 mL/m^2).[6,7] In the normal adult, RV mass is also only about one-sixth that of LV mass.[1] In childhood, there is a progressive regression of RV hypertrophy as pulmonary vascular resistance (PVR) decreases.

Traditionally, the RV has been divided into two components: the sinus (inflow) and the conus (infundibulum). The RV sinus extends from the tricuspid valve (inflow region) and includes the trabeculated (apical) portion of the ventricle (Fig. 1). The RV conus is usually free of muscular trabeculations and extends from the septomarginal band to the pulmonary valve (arterial trunk). In the anatomic LV, subaortic conal absorption occurs, which explains the absence of an infundibular portion.[1] Three prominent muscular bands divide the RV: the parietal, the septal and the moderator band. The parietal band and the infundibular septum make-up the crista supraventricularis, which separates the sinus and the conus regions.[8] The moderator band extends from the base of the anterior papillary muscle to the ventricular septum.[8]

In the study of complex congenital heart disease (CHD), it may be more useful to divide the RV into three parts: an inflow region, the trabeculated apical

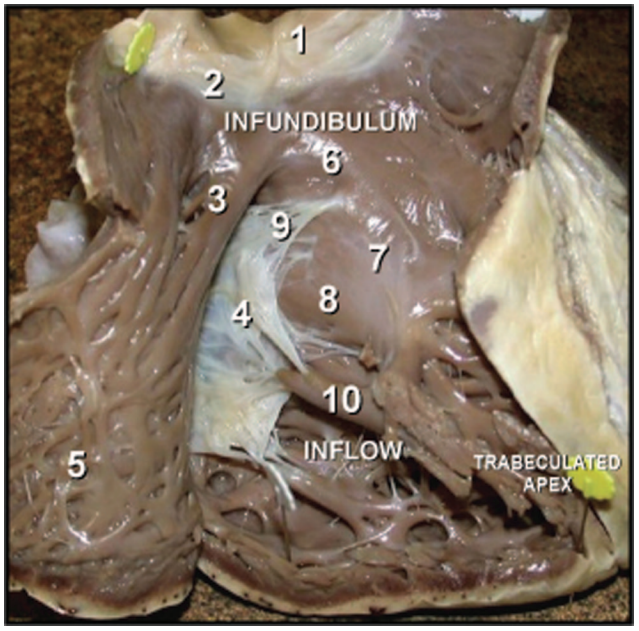


Figure 1. Right ventricular anatomy. The three regions: the inflow, the trabeculated apex, and the infundibulum are shown with detailed anatomical aspect. 1, pulmonary valve; 2, pulmonary annulus; 3, crista supraventricularis; 4, tricuspid valve; 5, right ventricular anterior wall; 6, anterior limb of trabecula septum marginalis (TSM); 7, body of TSM; 8, posterior limb of TSM; 9, medial papillary muscle; 10, anterior papillary muscle. Adapted with permission from Denault et al.[70]

myocardium, and the outflow region (infundibulum) (Fig. 1).[9] In hearts with congenital malformations, one or more of the three components may be rudimentary or absent.[9] Table 1 summarizes key anatomical and physiological features of the RV and LV.

RV PHYSIOLOGY

The primary function of the RV is to receive systemic venous return and pump it into the pulmonary system. Under normal conditions the RV, in contrast to the LV, is coupled to a low pressure, highly distensible arterial system.[1,3]

The RV is normally connected in series with the LV. In the absence of shunt physiology or significant valvular regurgitation, the stroke volume of the RV will normally match that of the left. Because of the greater end-diastolic volume of the right ventricle, RV ejection fraction (RVEF) is lower than the left. The lower limit of normal RVEF ranges from 40% to 45% compared with 50%–55% for LV ejection fraction.[7,10–12] Several mechanisms contribute to RV ejection, the most important being the bellows-like inward movement of the free wall. Other important mechanisms include the contraction of the longitudinal fibers, shortening of the long axis, drawing the tricuspid annulus toward the apex, and the traction on the free RV wall at its points of attachment to the LV as a result of LV contraction.[5] In contrast to the LV, twisting and rotational movements do not contribute significantly to RV contraction.[13] Furthermore, the contraction of the RV is also sequential, starting with the trabeculated myocardium and ending with the contraction of the infundibulum (normally separated by approximately 25–50 ms).[1,14]

To better understand the complex relationship between RV contractility, preload, and afterload, many investigators have studied the pressure-volume relationship of the RV (Fig. 2). One of their major findings was that the RV follows a time-varying elastance model in which ventricular elastance is described by the relationship between systolic pressure and volume under variable loading conditions.[14–16] Many studies have shown that RV elastance may also be approximated by a linear relationship.[1]

RV maximal elastance is considered by many investigators to be the most reliable index of RV contractility.[1] RV systolic elastance is lower than that of the LV. This arrangement implies that the RV is far more sensitive to increases in afterload.[16,17] This can be illustrated in the acute setting, where RV stroke volume decreases significantly after an increase in pulmonary arterial pressure (Fig. 3).[1,3,18]

The pulmonary circulation is an important determinant of RV afterload. The pulmonary vascular bed is a highly compliant, low-pressure, low-resistance system. In the presence of normal pulmonary circulation, the RV performs approximately one-fourth of the LV stroke work.[5] Several factors modulate PVR, including hypoxia, hypercarbia, cardiac output, pulmonary volume and pressure, and specific molecular pathways, most prominent being the nitric oxide pathway (vasodilation), the prostaglandin pathway (vasodilation), and the endothelin pathway (vasoconstriction).[1,3,19] Pulmonary vessels constrict with hypoxia (Euler-Liljestrand reflex) and relax in the presence of hyperoxia.[20] In some instances, hypercarbia may also be a strong pulmonary vasoconstrictor.

Lung volumes have a differential effect on intra- and extraalveolar vessels which accounts for the unique U-shaped relationship between lung volume and PVR. PVR is minimal at functional residual capacity and increased at large and small lung volumes alike (Fig. 4), this may be observed clinically when hyperinflation of the lungs greatly increases PVR.[20] Application of high levels of positive end-expiratory pressure may narrow the capillaries in the well ventilated lung areas and divert flow to less well ventilated or nonventilated areas, potentially leading to hypoxia. An increase in cardiac output distends open vessels and may recruit previously closed vessels decreasing PVR. Regional blood flow to the lungs is also influenced by gravity, where pulmonary blood flow is greater in the dependant areas of the lung.

Ventricular interdependence refers to the concept that through direct mechanical interactions the size, shape, and compliance of one ventricle may affect the size, shape, and pressure-volume relationship of the other.[21] The main anatomical determinants for

Table 1. Characteristics of Right and Left Ventricle

Characteristics	Right ventricle	Left ventricle
Structure	Inflow region, trabeculated myocardium, infundibulum	No infundibulum mitro-aortic continuity
Shape	From the side: triangular cross-section: crescentic	Elliptic
Volume (end-diastolic)	49–101 mL/m^2	44–89 mL/m^2
Mass (g/m^2)	<35 g/m^2 ≈ 1/6 LV mass	<130 g/m^2 (men) <100 g/m^2 (women)
Ejection fraction	40%–68% > 45%[a]	57%–74% > 50%[a]
Ventricular elastance (mm Hg/mL)	1.30 ± 0.84	5.48 ± 1.23
Ventricular compliance	Higher compliance than LV	$5.0 \pm 0.52 \times 10^{-2}$
Adaptation to disease	Better adaptation to volume overload states	Better adaptation to pressure overload states

Normal variables of the right (RV) compared with the left ventricle (LV). Range of normal values depends on method of acquisition.
[a] Lower value of normal RV and LV ejection fraction used in clinical practice.[1,5,7,10,11,16,66–68] Adapted from Haddad et al.[3]

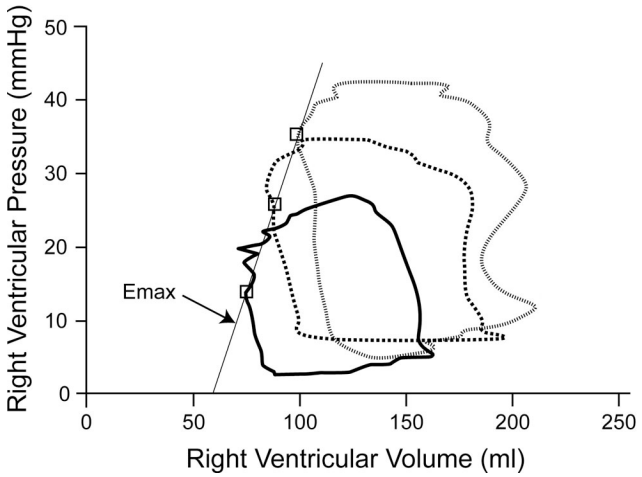


Figure 2. Pressure-volume loops of the right ventricle under different loading conditions. The slope of maximum time-varying elastance (E_{max}) is displayed on the graph. Adapted from Dell'Italia et al.[16]

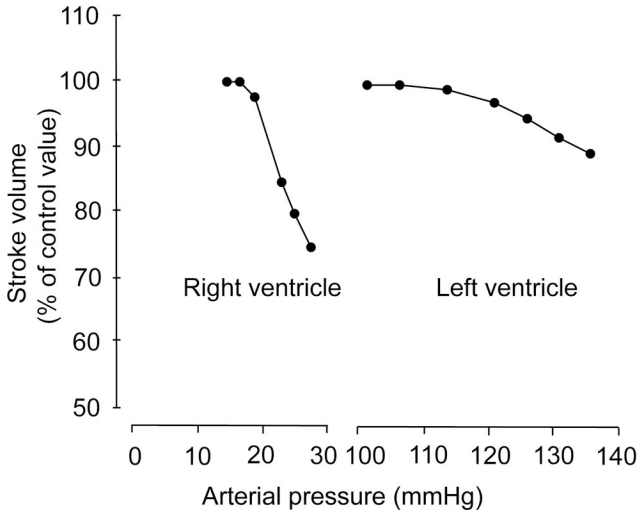


Figure 3. The response of the right and left ventricle to experimental increase in pressure or afterload. Adapted from MacNee et al.[18]

ventricular interdependence include the ventricular septum, the pericardium, and continuity between myocardial fibers of the RV and LV. Ventricular interdependence may occur in both systole and

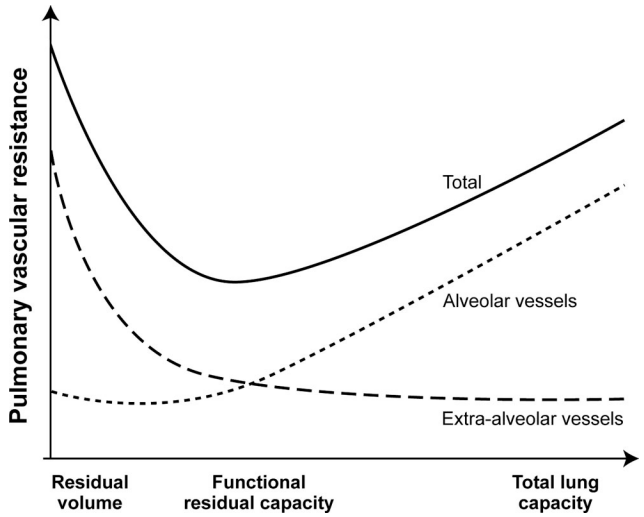


Figure 4. Relationship between lung volume and pulmonary vascular resistance (PVR). As lung volume is reduced or increased, the increase in PVR result from compression of the alveolar and extraalveolar vessels. RV = residual volume; FRC = functional residual capacity; TLC = total lung capacity. Adapted from Fischer et al.[20]

diastole. Although always present, ventricular interdependence is most evident with changes in loading conditions, such as those observed during respiration or sudden postural changes.[1,3,21] Ventricular interdependence also plays an important part in the pathophysiology of RV dysfunction.

PERIOPERATIVE ASSESSMENT OF THE RV

Overview

In cardiac surgery, right heart catheterization and echocardiography play an essential and complementary role in the assessment of RV structure and function. Both techniques provide useful information that may help tailor the anesthetic and surgical approach and provide guidance in the management of hemodynamically unstable patients. Hemodynamically, RV dysfunction or failure is usually recognized in the presence of a right atrial pressure (RAP) ≥8–10 mm Hg or a RAP to pulmonary capillary wedge pressure ≥0.8 (isolated RV failure) and/or a low cardiac index ($\leq 2.2\ L \cdot min^{-1} \cdot m^{-2}$).[22,23] Increasing RAP may also

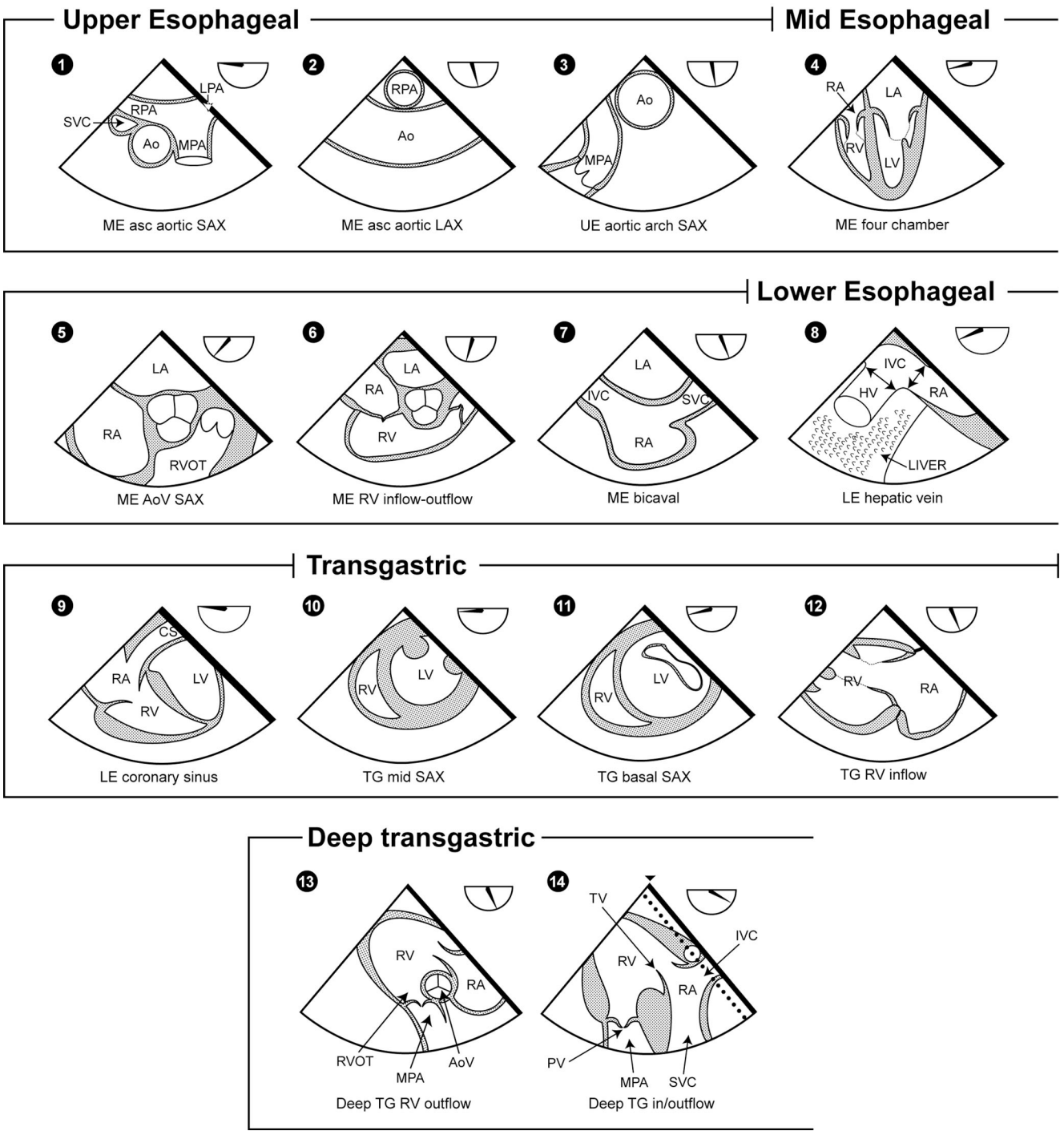


Figure 5. Upper esophageal (UE), mid-esophageal (ME), low esophageal (LE), transgastric (TG), and deep TG views useful in the evaluation of right ventricular (RV) function. Asc = ascending; Ao = aorta; AoV = aortic valve; CS = coronary sinus; IVC = inferior vena cava; LA = left atrium; LAX = long axis, LPA = left pulmonary artery; LV = left ventricle; MPA = main pulmonary artery; PV = pulmonic valve; RA = right atrium; RPA = right pulmonary artery; RV = right ventricle; RVOT = right ventricular outflow tract; SAX = short axis; SVC = superior vena cava; TV = tricuspid valve. Adapted from Shanewise et al. and Denault et al.[24,71]

be a sign of impeding RV failure. Significant RV outflow tract (RVOT) obstruction may also be suspected in the presence of RV-pulmonary artery gradient more than 25 mm Hg. As will be reviewed in this section, echocardiography also provides useful information on RV and pulmonary structure, valvular function and pericardial physiology. Four recently published guidelines may help to guide the echocardiographic assessment of the RV: 1) ASE/SCA (American Society of Echocardiography/Society of Cardiovascular Anesthesiologists) guidelines for performing a comprehensive intraoperative multiplane transesophageal echocardiography (TEE) examination; 2) ASE/SCA guidelines for a comprehensive epicardial echocardiography examination; 3) the ASE and European Association of Echocardiography guidelines on

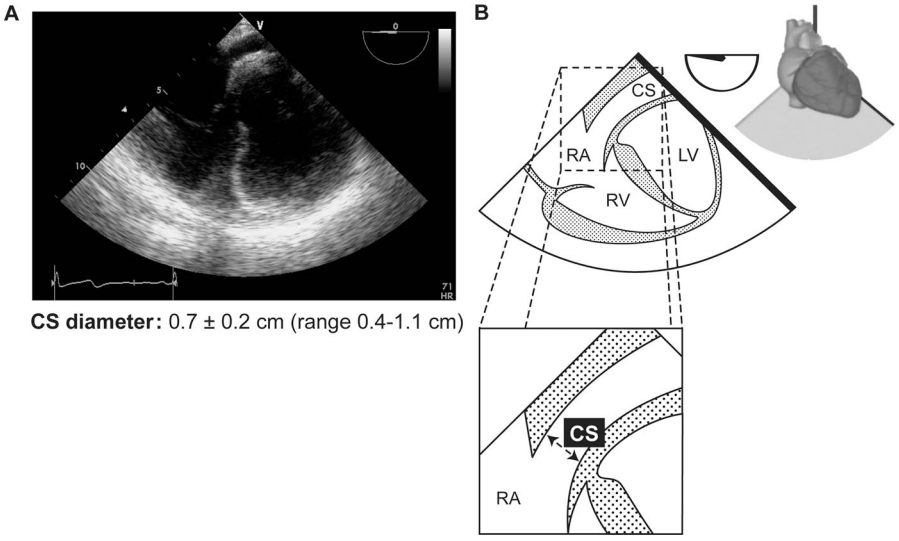


Figure 6. Lower esophageal four-chamber view of the coronary sinus (CS). The diameter is measured 1 cm proximal to where it enters the right atrium (RA).[69] LV = left ventricle; RV = right ventricle. Reproduced with permission from Denault et al.[70]

two-dimensional (2D) chamber quantification; and 4) the ASE recommendations for evaluation of the severity of native valvular regurgitation with 2D and Doppler echocardiography.[12,24–26]

Echocardiographic Views of the RV

In the operating room, both TEE and epicardial echocardiography are helpful in obtaining images of the RV. In the intensive care unit, TEE and transthoracic echocardiography (TTE) constitute useful modalities.

The most useful transesophageal views are illustrated in Figure 5.[24] The midesophageal four-chamber view is ideal for visualizing the RV lateral wall and measuring RV internal dimensions and RV fractional area change (RVFAC) (Videos 1 and 2; please see video clips available at www.anesthesia-analgesia.org). The midesophageal views are also useful in visualizing the coronary sinus (Fig. 6), assessing tricuspid regurgitation (usually achieved at an angle of 30°–60°), and assessing potential atrial or ventricular septal defects. The transgastric views allow short-axis views (SAX) of the RV and septum, and views of the RV inflow tract and RVOT, inferior vena cava (IVC) as well as hepatic veins. The anterior and inferior walls of the RV are best visualized in the transgastric views. The great vessels are best studied in the upper esophageal views, whereas tricuspid annular tissue Doppler signals are best assessed in the deep transgastric RV views.

Epicardial echocardiography may be very helpful in the presence of a contraindication to TEE such as an esophageal stenosis, if the images obtained by TEE are suboptimal, for diagnosis of pulmonic valve pathology or for detection of intraoperative thromboembolism.[27,28] Most of the imaging planes from TEE can be obtained using epicardial echocardiography (Fig. 7). However, the four recommended ASE views which provide helpful images of the RV include 1) the epicardial aortic valve (AoV) SAX view (TTE parasternal AoV SAX equivalent) (Fig. 7B, Video 3; please see video clips available at www.anesthesia-analgesia.org); 2) the epicardial LV long-axis (LAX) view (TTE parasternal LAX equivalent) (Fig. 7C, Video 4; please see video clips available at www.anesthesia-analgesia.org); and 3) the epicardial LV basal SAX view (TTE modified parasternal mitral valve basal SAX equivalent) (Fig. 7D); and 4) the epicardial RVOT view (TTE parasternal SAX equivalent) (Fig. 7E). Guidelines on this topic were published in 2007.[26]

Challenges in the Echocardiographic Study of the RV

The echocardiographic study of the RV is more challenging than that of the left. The main difficulties encountered may be explained by 1) the complex shape of the RV, 2) heavy apical trabeculations of the RV, which limits endocardial surface recognition, and 3) the marked load dependence of several indices of RV function.[5] Despite these limitations, a comprehensive assessment of the RV may provide important insights into its contractility, preload, and afterload.

Identifying the Anatomic RV

Although the RV is usually on the right side of the heart and connects with the pulmonary artery, the anatomic RV is defined by its structure not by its position or connections. Features which help differentiate the anatomic RV from anatomic LV include 1) the more apical insertion point of the septal leaflet of the tricuspid valve relative to the anterior leaflet of the mitral valve, 2) the presence of a moderator band, 3) the presence of more than 2 papillary muscles, and 4) the trileaflet configuration of the tricuspid valve.[1,29,30] This is especially important in CHD, where the anatomic RV may be positioned on the left side of the heart or connect to the aorta.[3,30] In a corrected transposition of the great vessels (L-TGA), the anatomic RV is positioned on the left side of the heart and connects to the aorta (systemic ventricle). In a D-transposition of great arteries (D-TGA), the anatomic RV is positioned

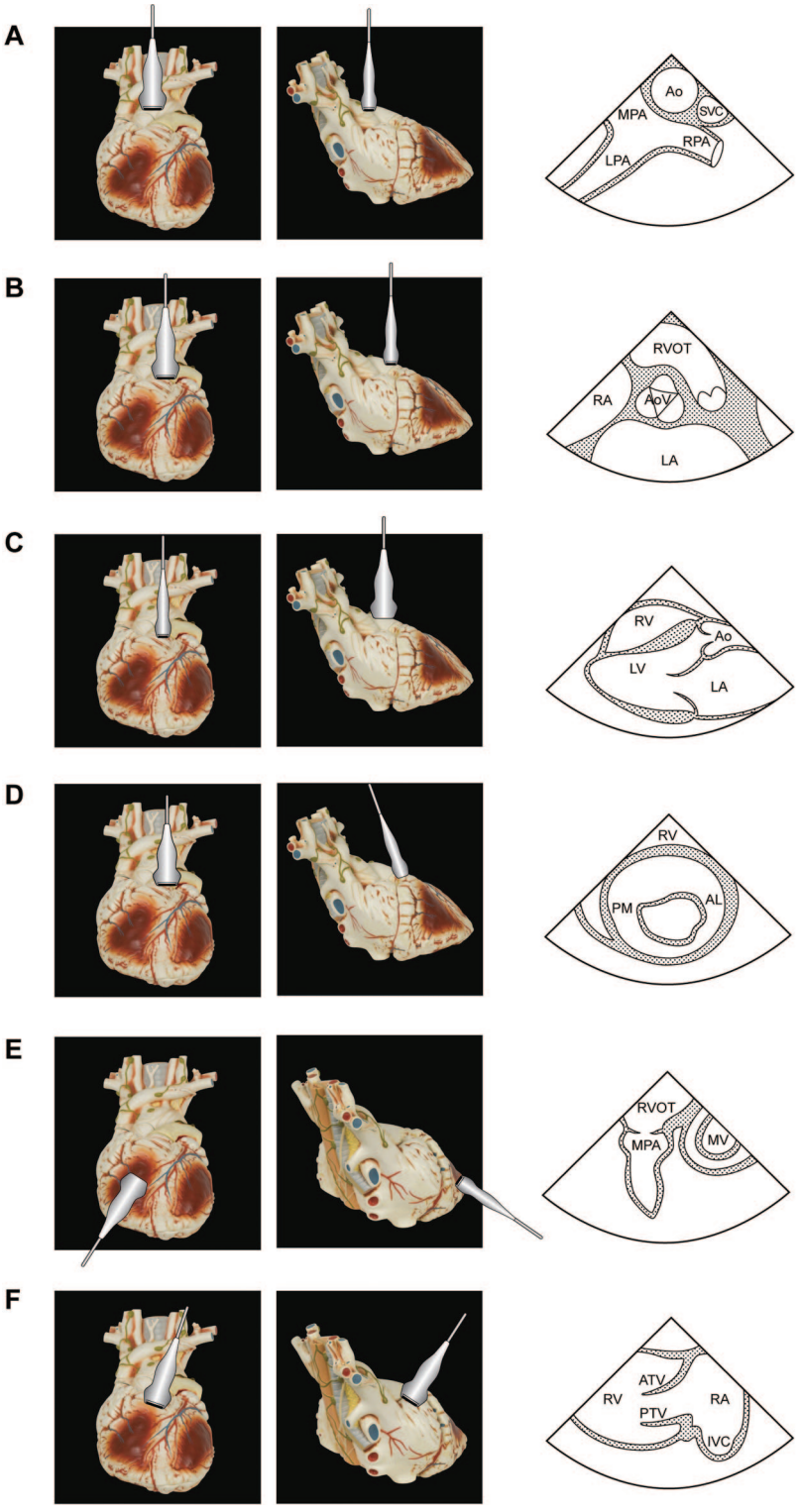
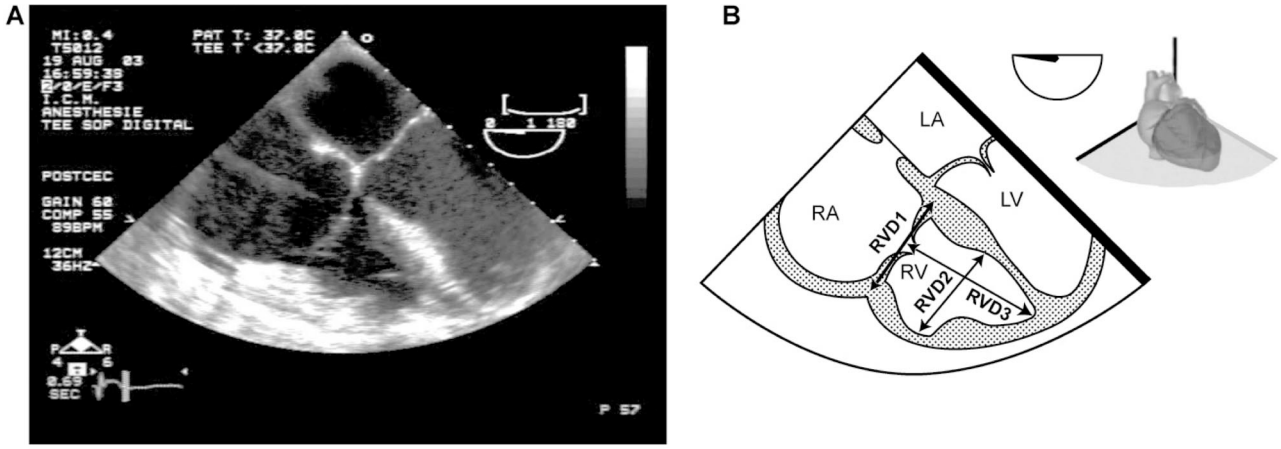


Figure 7. Epicardial views of the right ventricle. (A) Main, left and right pulmonary artery views; (B) Aortic valve (AoV) short axis (SAX) view (Video 3; please see video clips available at www.anesthesia-analgesia.org); (C) Left ventricular long-axis (LAX) view (Video 4; please see video clips available at www.anesthesia-analgesia.org); and (D) Left ventricular basal SAX view E. Right ventricular outflow tract view F. Right atrial and ventricular views. Figure B–E are the recommended views[26] but views (A) and (E) can be used to complete the evaluation of the right ventricle. AL = anterolateral; Ao = aorta; AoV = aortic valve; ATV = anterior tricuspid leaflet; CS = coronary sinus; IVC = inferior vena cava; LA = left atrium; LPA = left pulmonary artery; LV = left ventricle; MPA = main pulmonary artery; MV: mitral valve; PM = posteromedial; PTV = posterior tricuspid valve; RA = right atrium; RPA = right pulmonary artery; RV = right ventricle; RVOT = right ventricular outflow tract; SVC = superior vena cava; TV = tricuspid valve.

to the right side of the heart and connects to the aorta. Alternatively, the LV may show more pronounced trabeculations, which may mimic the structure of the anatomic RV (noncompaction of the LV).

RV Size and Shape

Because the complex shape of the RV does not lend itself to simple mathematical modeling, the assessment of RV size using 2D echocardiography remains challenging. The best correlations between 2D echocardiography and RV volumes have been obtained using the maximal SAX dimension and the RV area measured in the four-chamber view (Fig. 8).[5,12,31] It is, however, important to note that there is significant overlap between normal patients and patients with RV volume overload.[12] Furthermore, normal 2D values have not been well established in patients requiring mechanical ventilation. The availability of 3D TEE



C

	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal
RVD1, cm	2.0 - 2.8	2.9 - 3.3	3.4 - 3.8	≥ 3.9
RVD2, cm	2.7 - 3.3	3.4 - 3.7	3.8 - 4.1	≥ 4.2
RVD3, cm	7.1 - 7.9	8.0 - 8.5	8.6 - 9.1	≥ 9.2

Figure 8. Mid-esophageal four-chamber view (A, B) with the specific recommended measurements (C). The right ventricular diameter (RVD) 1 correspond to the tricuspid annulus, RVD2 to the minor axis and RVD3 to the major axis. BSA = body surface area; LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle; RVD = right ventricular diameter. Adapted from Lang et al.[12]

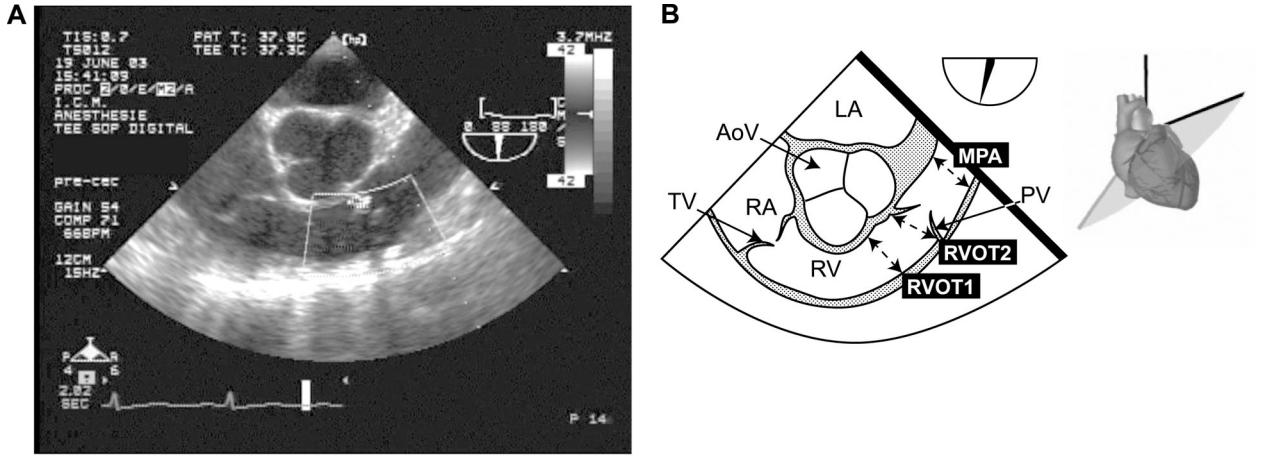
technology may in the future allow better intraoperative assessment of RV volumes. At this time, however, software used for RV volume quantification is not routinely available. Figure 9 illustrates normal values of RVOT measurements.

RV hypertrophy is echocardiographically defined as a ventricular wall thickness more than 5 mm at end-diastole. The inferior or lateral walls of the RV are the preferred locations for measurement since, in contrast to the anterior wall, they are not invested with as much epicardial fat.[32] The inferior wall of the RV is best assessed in transgastric views performed at 0°, whereas the RV lateral wall is best measured in the four-chamber view. Because the RV wall is thinner and more trabeculated than the LV wall, precise measurements are more difficult to obtain.

The RV shape cannot be described by one simple geometric shape. Under normal conditions, the RV appears triangular, when viewed from the side and crescentic in cross-section.[5] The analysis of septal curvature may provide useful insights into RV pathology. The interventricular septum is usually curved (convex) toward the RV (Fig. 10). The eccentricity index, a measure of septal curvature, represents the ratio of the LV minor axis diameter (parallel to the septum) to its perpendicular axis.[33,34] In normal subjects, the index is essentially one at both end-diastole and end-systole. In the adult with acquired pressure overload, the RV dilates early and the ventricular septum is displaced toward the LV cavity especially at end-systole. This will distort both RV and LV geometry (D-shaped LV; eccentricity index >1).[30] In volume overloaded states, the RV is dilated and rotated clockwise (apical reference). The RV initial crescentic shape is transformed into a more cylindrical configuration, and the ventricular septum is displaced toward the LV cavity (D-shape LV; eccentricity index >1), mainly at end-diastole.[30] In patients with congenital pulmonary stenosis, the RV has a greater hypertrophic response, and its shape is more elliptic; dilation occurs late in the course of the disease or in the presence of a critical stenosis. When aneurysms are seen in the RV, the possibility of arrhythmogenic RV dysplasia must be considered. In this condition, the aneurysms occur most commonly in the anterior infundibulum, basal inferior wall, and apex. Other causes of RV aneurysms include myocardial infarction and, in rare cases, absence of a right pericardium.[35,36]

Indices of RV Function

The study of RV function comprises indices that reflect RV systolic function, RV diastolic function, global and regional RV function, (systole and diastole) and valvular function.[37,38] The study of RV dyssynchrony is a new field of research and could play a larger role in the future. Indices of RV systolic function may describe the extent of RV contraction or reflect RV contractility (i.e., the intrinsic ability of the ventricle to contract). An ideal index of contractility would be independent of afterload and preload, sensitive to change in inotropic state, independent of



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	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal
RVOT1, cm	2.5 - 2.9	3.0 - 3.2	3.3 - 3.5	≥ 3.6
RVOT2, cm	1.7 - 2.3	2.4 - 2.7	2.8 - 3.1	≥ 3.2
MPA, cm	1.5 - 2.1	2.2 - 2.5	2.6 - 2.9	≥ 3.0

Figure 9. Mid-esophageal right ventricular inflow and outflow view at 45°. The ventricular measurements are obtained during systole and the main pulmonary artery (MPA) measurement is obtained during diastole. Both the two-dimensional images and the electrocardiogram are used to identify systole and diastole (A, B). The recommended measurements and reference ranges are shown (C). (AoV = aortic valve; LA = left atrium; PV = pulmonic valve; RA = right atrium; RV = right ventricle; RVOT = right ventricular outflow tract; TV = tricuspid valve).[12]

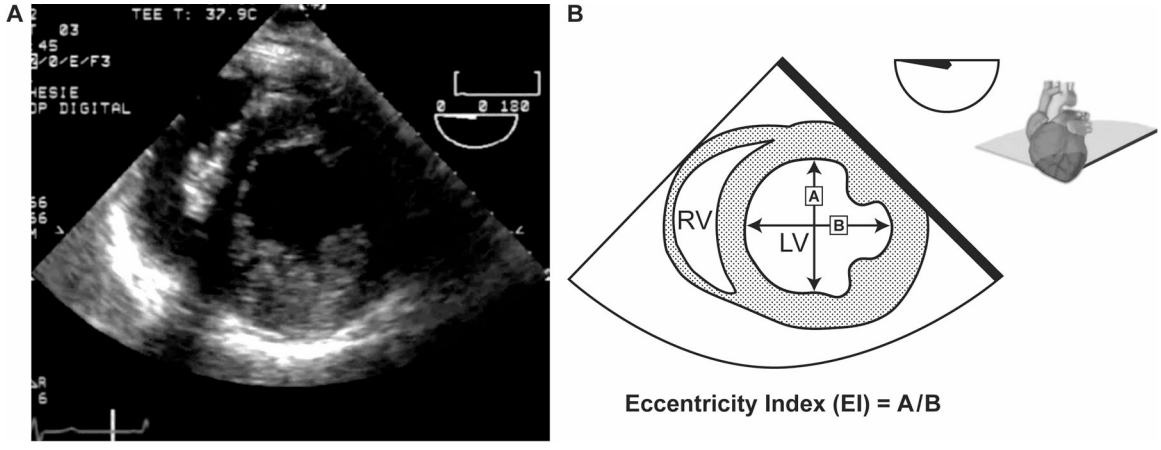

Figure 10. Transgastric mid-papillary view of the left ventricle (LV) and right ventricle (RV). The eccentricity index (EI) corresponds to the ratio of the vertical (A)/horizontal (B) diameter of the LV.

heart size and mass, easy and safe to apply, and proven to be useful in the clinical setting.[39] The most commonly used echocardiographic indices of RV systolic function are summarized in Tables 2 and 3 and include

1. Geometric indices, those which reflect the extent of contraction, such as RVFAC, RVEF, and tricuspid annular plane systolic excursion (TAPSE).
2. Myocardial velocity indices, such as the tricuspid annular plane maximal systolic velocity and the isovolumic acceleration (IVA).
3. Hemodynamic indices, such as the RV first derivative of pressure and time (RV dP/dt).
4. Time interval indices, such as the RV myocardial performance index (RVMPI) or Tei index which reflect both systolic and diastolic parameters.

Indices of RV Systolic Function

RVEF represents the ratio of stroke volume to end-diastolic RV volume ([RVEDV-RVESV]/RVEDV). RVEF has the advantage of being a widely accepted and validated index of RV function. Its prognostic value has been proven in heart failure, valvular heart disease, and CHD.[3] RVEF has, however, the disadvantage of being highly load dependent and may not always reflect ventricular contractility in volume or

Table 2. Markers of Right Ventricular Dysfunction Associated with Clinical Status and Prognosis

1. Systolic performance indices
   a. RVEF
   b. RVFAC
   c. TAPSE (tricuspid annular plane systolic excursion)
2. Diastolic profile indices
3. RV myocardial performance index
4. Right-sided dilatation
   a. RV dilatation absolute or relative to left ventricle
   b. Right atrial size
5. Hemodynamics
   a. Right atrial pressure
   b. Cardiac index
   c. dP/dt_{max}
6. Ventricular elastance and pressure-volume or pressure-area loops
7. Tissue Doppler or strain indices
8. Tricuspid regurgitation
9. Electrophysiological characteristics
   a. Heart rate variability
   b. Arrhythmias
10. Neurohormones and cytokines
   a. B-type natriuretic peptide
   b. Norepinephrine
   c. Endothelin
   d.Tumor necrosis factor

RV = right ventricular; RVEF = right ventricular ejection fraction; RVFAC = right ventricular fractional area change; TAPSE = tricuspid annular plane systolic excursion. Adapted from Voelkel et al. and Haddad et al.[37,38]

pressure overloaded states. An accurate assessment of RVEF using echocardiography also remains difficult because of the complex shape and heavy trabeculations of the RV. In 2D echocardiography, RVEF may be assessed using Simpson's rule or the area length method.[40] Because of the complex geometry of the RV and limited RV endocardial definition, reliable estimates of RVEF using 2D echocardiography remain elusive. In the future, 3D acquisition protocols may provide more accurate assessments of RV volume and RVEF.[41]

Echo-derived RVFAC is an index of RV systolic function that is easier to measure. RVFAC represents the ratio of RV systolic area change to the end-diastolic area. It is measured in the four-chamber view and can be systematically incorporated into a basic echocardiographic study (Fig. 11). In nonsegmental disease, a good correlation has been reported between RVFAC and RVEF measured using magnetic resonance imaging.[42] A consensus from the American and European Societies of Echocardiography has determined the ranges in the evaluation of RVFAC: normal values are between 32% and 60%, mildly abnormal between 25% and 31%, moderately abnormal between 18% and 24%, and severely abnormal below 17%.[12]

TAPSE measures the longitudinal systolic motion of the free edge of the tricuspid valve annulus.[43] It is measured using M-mode imaging in the four-chamber view,[44] typically on the lateral annulus, although some authors have used the inferior annulus and obtained similar values[45] (Fig. 11, Videos 5a and 5b; please see video clips available at www.anesthesia-analgesia.org). Most of the studies using TAPSE were done using TTE. Compared with RVEF and RVFAC, TAPSE has the advantage of not being limited by RV endocardial border recognition. Preoperatively, it may represent a reasonable index of global systolic function; however, its reliability postoperatively has not been as well established.[3]

Systolic tissue Doppler velocity of the tricuspid annulus (St) has been studied as an index of RV function using both spectral pulsed wave tissue Doppler and color tissue Doppler (Fig. 12, Video 6; please see video clips available at www.anesthesia-analgesia.org). In patients with heart failure, a moderate correlation was noted between St velocity and RVEF ($r = 0.65$, $P < 0.001$).[46] Currently, its predictive value in cardiac surgery is not well established.

The IVA is a recently described index of systolic performance that is relatively load independent. It is calculated by dividing the maximal isovolumic myocardial velocity by the time to peak velocity using spectral pulsed wave or color tissue Doppler (Fig. 13). In 2002, Vogel et al. used color tissue Doppler to study tricuspid annular IVA in a closed chest animal model during modulation of preload, afterload, contractility, and heart rate. Their study demonstrated that, of all myocardial velocity parameters, IVA was the most reliable noninvasive index of contractility. Three clinical studies confirmed its value in CHD, i.e., postrepair of tetralogy of Fallot (TOF), TGA, and after cardiac surgery.[47–50] Further validation of this promising new index in cardiac surgery is, however, required.

The maximum first derivative of RV pressure development (dP/dt max) has also been used as an index of RV contractility. This index may be calculated using continuous-wave Doppler of the tricuspid valve and the Bernoulli equation to calculate the pressure difference from 1 m/s to 2 m/s. Obtaining a reliable signal of tricuspid regurgitation in TEE, however, may be more difficult than in TTE. Furthermore, it has been demonstrated, in numerous studies, that RV dP/dt_{max} is significantly affected by loading conditions and cannot be used as a reliable index of contractility.[51] It may, however, be useful in assessing directional changes in response to therapy, assuming stable loading conditions.

The RVMPI has been described as a nongeometric index of global ventricular function (Fig. 14).[52] It represents the ratio of isovolumic time intervals to ventricular ejection time (ET) and is calculated as MPI = (IVCT + IVRT)/ET, where IVCT is the isovolumic contraction time, and IVRT is isovolumic relaxation time.[52] RVMPI increases in the presence of systolic or diastolic dysfunction. RVMPI has been validated in several disease states, including CHD, primary pulmonary hypertension, myocardial infarction, and chronic respiratory disease.[53–56] A small prospective study has suggested that RVMPI may be useful in stratifying patients undergoing high-risk valvular surgery.[38] It is important to remember that RVMPI is less reliable in the presence of arrhythmias

Table 3. Functional Variables of the Right Ventricle

Functional parameters	Normal value	Load dependency[a]
Systolic performance variables		
RVFAC (%)	32%–60%	+++
RVEF (%)	45%–68%	+++
TAPSE	>15 mm	+++
Tricuspid annular plane maximal systolic velocity (using spectral pulsed wave tissue Doppler)	>12 cm/s	++
IVA (using tissue pulsed wave Doppler)	1.4 ± 0.5 m/s^2	+
Diastolic parameters		
IVC dimension (cm), collapse index	<1.7 cm, CI >50%	+++
Tricuspid early (E) to late (A) filling velocity ratio	1.5 ± 0.3	+++
Hepatic vein profile (S: systolic, D: diastolic)	S/D velocity ratio >1, no S reversal, atrial reversal <50% S	+++
IVRT	<60 ms	+++
Rapid myocardial filling velocity (E_t) (cm/s)	E_t: 15.6 ± 3.9	+++
Late diastolic myocardial filling velocity, A_t (cm/s)	A_t: 15.4 ± 4.5	+++
Combined systolic and diastolic parameter		
RVMPI	0.28 ± 0.04	++

The severity of RV systolic dysfunction may be graded using RVFAC and RVEF. Using RVFAC, mild dysfunction: 25%–31%, moderate dysfunction: 18%–24%, severe dysfunction <17%; Using RVEF, mild dysfunction: 35%–44%, moderate dysfunction: 26%–34%, severe dysfunction <25%. Almost all normal values have been established in nonventilated patients.

[a] Refers to the degree of load dependency from minimal + to significant +++. IVA = isovolumic acceleration using Doppler tissue imaging; IVC = inferior vena cava; RVEF = right ventricular ejection fraction; RVFAC = right ventricular fractional area change; RVMPI = right ventricular myocardial performance index; TAPSE = tricuspid annular plane systolic excursion; St = maximal systolic tricuspid annular plane velocity; IVRT = isovolumic relaxation time.

Adapted from Dell'Italia,[1] Leng,[5] Lorenz et al.,[7] Hurwitz et al.,[10] Pfisterer et al.,[11] Santamore et al.,[21] Weyman,[29] Davlouros et al.,[30] Tei et al.,[52] Cohen et al.[69]

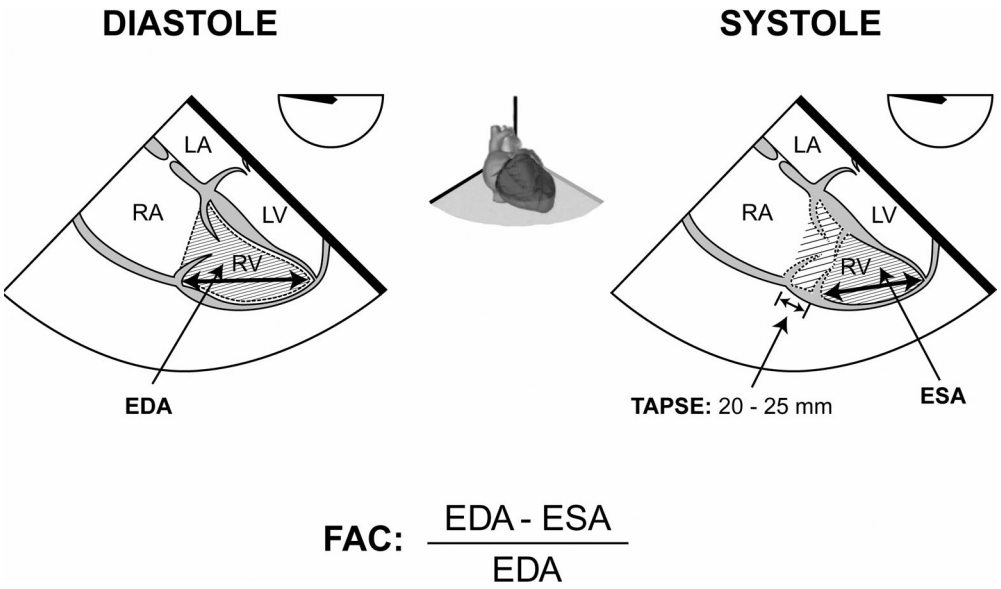

Figure 11. Measurement of the right ventricular fractional area change (FAC) and tricuspid plane systolic annular excursion (TAPSE). The FAC corresponds to the difference of the right ventricular end-diastolic area (EDA) minus the right ventricular end-systolic area (ESA) divided by the right ventricular EDA. The TAPSE is another technique to quantify right ventricular function. It represents the systolic excursion of the lateral tricuspid annulus and can be measured from the change in the distance (arrow) in diastole compared with diastole between the apex and the lateral tricuspid annulus. Normal TAPSE should be 20–25 mm. LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.

or high-grade atrioventricular block. Pseudonormalization of the RVMPI has also been reported in acute, severe RV myocardial infarction.[56]

Regional RV Systolic Function

The pattern of regional RV dysfunction may also be helpful in differentiating causes of RV dysfunction. For example, in pulmonary embolism, McConnell et al.[57] described a distinct pattern of RV dysfunction characterized by severe hypokinesis of the RV mid-free wall associated with normal contraction of the apical segment (best seen in the four-chamber view). In contrast, in other causes of pulmonary hypertension, apical contraction is often depressed.

Variables of RV Diastolic Function

RV diastolic function has not been as extensively studied as that of the LV. Clinically useful diastolic

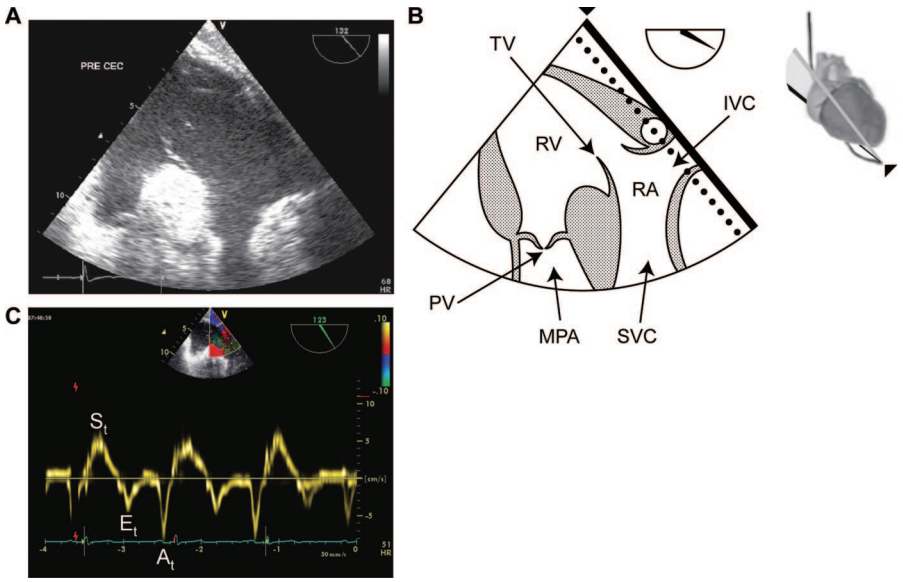

Figure 12. (A) Deep transgastric right ventricular in/outflow long-axis view. This view allows the evaluation of both the pulsed wave Doppler interrogation of the tricuspid valve and tissue Doppler imaging of the tricuspid annulus along the dotted line. (B) The darker line on the right side of the triangular sketch is matched to the thicker line on the triangular slice of the three-dimensional (3D) icon. (C) Tissue Doppler signal obtained at the base of the tricuspid annulus (Video 6; please see video clips available at www.anesthesia-analgesia.org). A_t = tricuspid late diastolic filling (during atrial contraction) tissue Doppler velocity; E_t = tricuspid early diastolic filling tissue Doppler velocity; IVC = inferior vena cava; MPA = mean pulmonary artery; PV = pulmonic valve; RA = right atrium; RV = right ventricle; St = tricuspid systolic tissue Doppler velocity; SVC = superior vena cava; TV = tricuspid valve. Adapted with permission from Denault et al.[71]

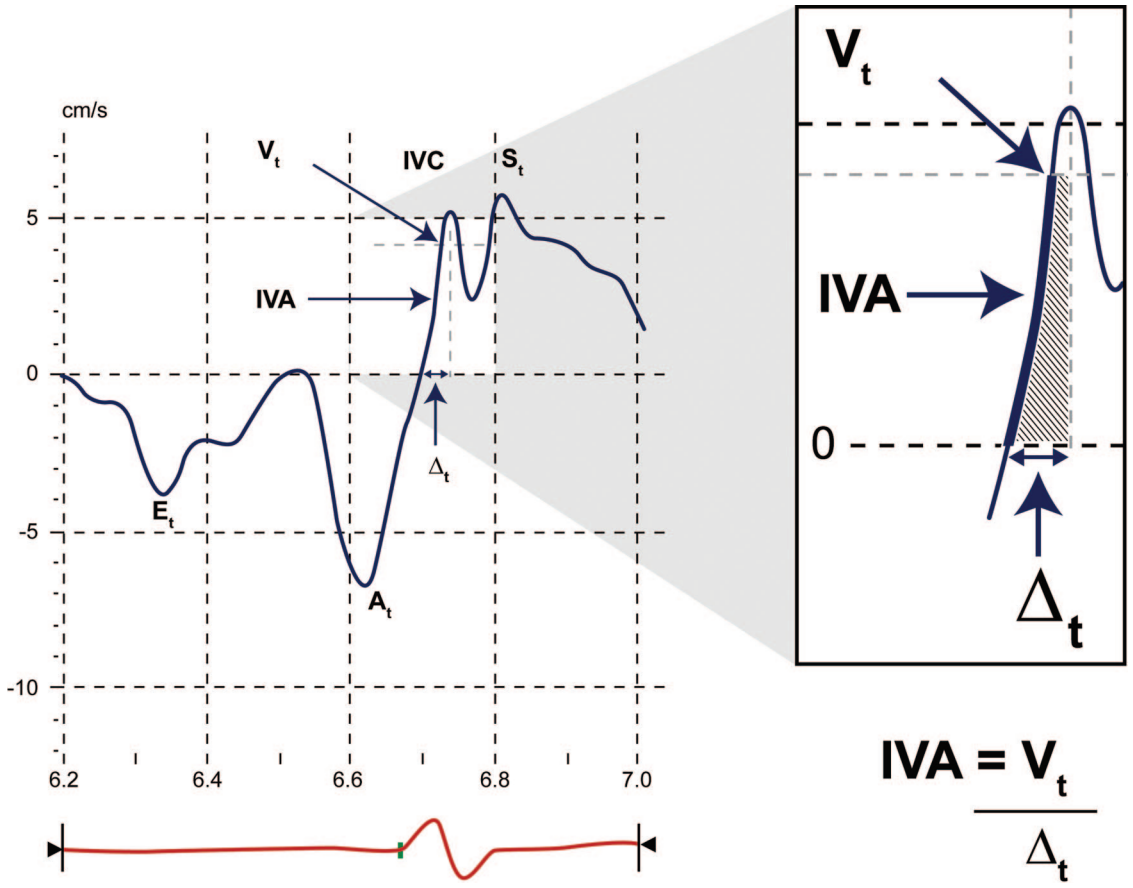

Figure 13. Mean tissue color Doppler velocities of the basal tricuspid annulus during a cardiac cycle. The dominant slope is used to measure the isovolumic acceleration (IVA).[50] A_t = tricuspid late diastolic filling (during atrial contraction) tissue Doppler velocity; E_t = tricuspid early diastolic filling tissue Doppler velocity; IVC = isovolumic contraction; St = tricuspid systolic tissue Doppler velocity; t = time; V_T = tissue Doppler velocity during IVC.

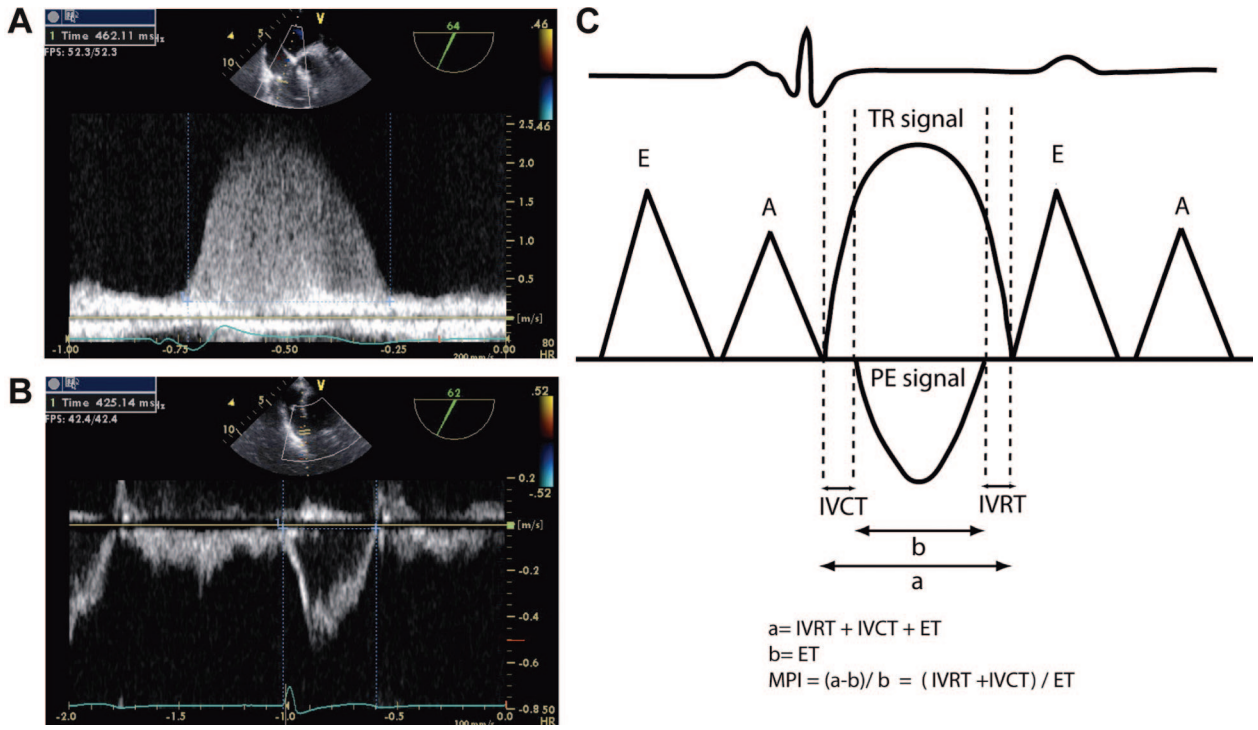

Figure 14. Measurement of the right ventricular myocardial performance index (RVMPI). The RVMPI is the difference between total time of tricuspid regurgitation (TR) or the time between A and E tricuspid flow signal (A,B) and the ejection time (ET), divided by the ET (C). The TR duration is obtained from a mid-esophageal inflow-outflow view (Fig. 5.4 or 5.6) and the ET measured from an upper-esophageal (Fig. 5.1 or 5.3) or deep transgastric in/outflow (Fig. 5.14). IVCT = isovolumic contraction time; IVRT = isovolumic relaxation time; E = tricuspid rapid filling velocity; A = atrial rapid filling velocity; PE = pulmonary ejection. Adapted from Haddad et al.[38]

variables of RV diastolic function include RAP, RV filling profiles, and hepatic vein profiles.[58] Compared with LV filling, the velocities across the tricuspid valve are significantly lower than those of the mitral. The tricuspid deceleration time is also longer than mitral deceleration time. Tricuspid filling profiles are usually measured in the mid-esophageal view or the transgastric view with rightward rotation of the probe.

In nonventilated patients, the IVC size and collapse index correlate well with RAP. The collapse index refers to the relative decrease in IVC diameter with inspiration (as with sniffing). An IVC size <2 cm with a collapse index of more than 50% usually corresponds to a RAP <5 mm Hg. A dilated IVC with a collapse index of $\leq 10\%$ usually corresponds to a RAP of 20 mm Hg. Although correlations in ventilated patients have not been as well validated, studies suggest that the collapse index percentage of the IVC correlates with fluid responsiveness.[59]

Only a few studies have assessed the importance of RV diastolic filling profiles in cardiac surgery.[58,60–62] In a study by Carricart et al.,[61] abnormal hepatic venous flow velocities before cardiac surgery were associated with an increased need for vasoactive support after cardiopulmonary bypass. These flow velocities, however, were not shown to be an independent predictor of worse outcome on multivariate analysis. In a study by Denault et al.[62], abnormal preoperative RV diastolic profiles were associated with difficult separation from cardiopulmonary bypass. Further studies are needed to validate these findings and to assess the independent value of RV diastolic function in cardiac surgery.

In TOF, a restrictive RV filling profile has been associated with worse outcome early after repair of TOF. A "restrictive RV physiology," as described in TOF, is characterized by the presence of forward and laminar late diastolic pulmonary flow throughout respiration.[30] In the presence of a noncompliant RV, atrial contraction results in an increase in RV pressures exceeding pulmonary pressures, thus resulting in late diastolic pulmonary flow. Early after TOF repair, a restrictive RV pattern, suggestive of a noncompliant ventricle, has been associated with a low cardiac output and longer intensive care stays.[30,63] Late after TOF repair, however, restrictive RV physiology counteracts the effects of chronic pulmonary regurgitation and is associated with a smaller RV, shorter QRS duration and increased exercise tolerance.[30,64]

Ventricular Interdependence

Ventricular interdependence may be exaggerated in patients with constrictive pericarditis or tamponade physiology, whereas it is usually in the normal range in restrictive disease. Other factors which may increase ventricular interdependence include loading conditions and increased intrathoracic pressures.[21]

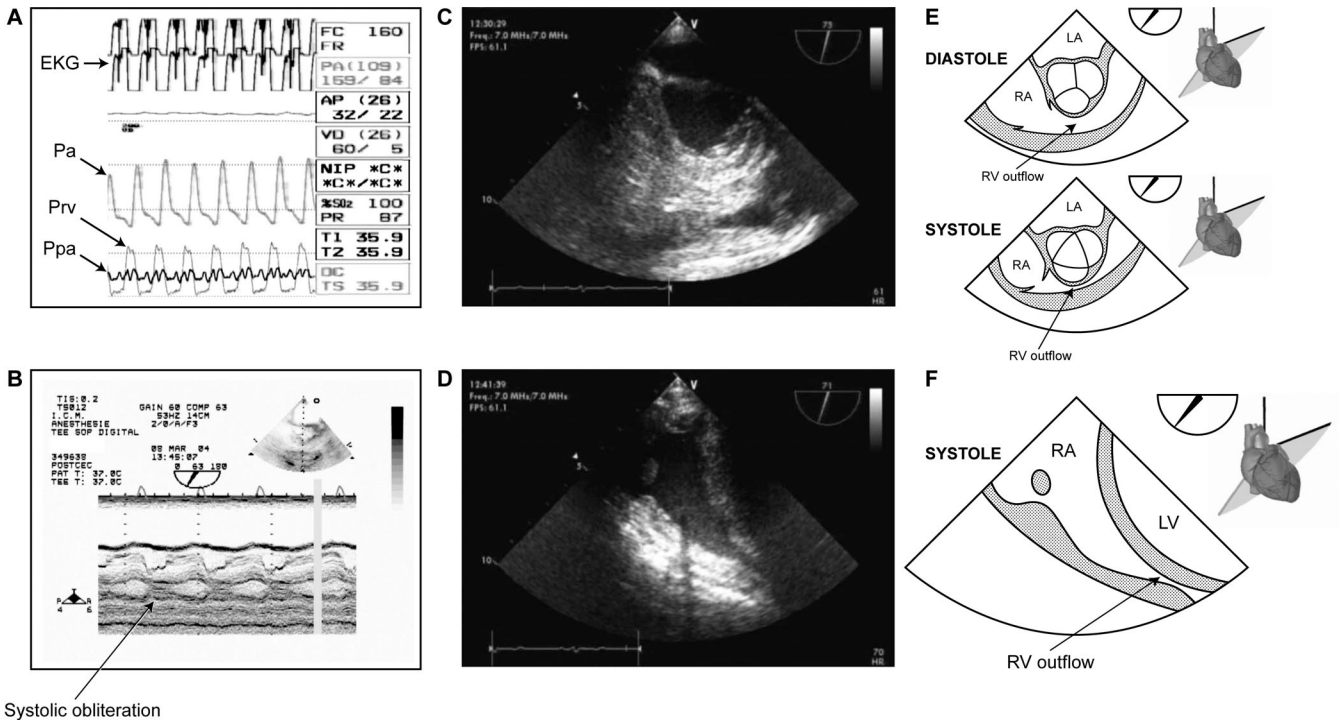

Figure 15. Dynamic right ventricular outflow tract obstruction in a 28-year-old man after aortic valve replacement. A 17 mm Hg gradient between the right ventricular pressure (Prv) and the pulmonary artery pressure (Ppa) is present. An M-mode view obtained in the mid-esophageal inflow-outflow view demonstrates the right ventricular outflow tract collapse (B). This is also shown in the same view during systole (C–E) and in a deep right ventricular outflow tract (D–F). EKG = electrocardiogram; LA = left atrium; LV = left ventricle; PA = arterial pressure; RV = right ventricle.

The two most useful features that suggest increased ventricular interdependence include the presence of increased reciprocal respiratory changes in tricuspid and mitral inflow maximal velocity and reciprocal respiratory changes in RV and LV size. These features have mainly been studied in nonventilated patients.

Under normal conditions and spontaneous respiration, the tricuspid inflow maximal velocity increase with inspiration is usually <15%, whereas that of the mitral valve is usually <10%. In constrictive pericarditis, the tricuspid velocity change is often higher than 40%, whereas mitral inflow velocity change with respiration is usually more than 25%. In tamponade, the tricuspid respiratory velocity change is often higher than 85% (increasing with inspiration), whereas that of the mitral valve is usually more than 40% (decreasing with inspiration).[65] In contrast, in restrictive physiology, the respiratory changes in tricuspid and mitral velocities are usually not increased and the early filling to atrial contraction ratio is often greater than 2 with a mitral inflow deceleration time of <150 ms. There is usually no abnormal septal motion. It is, however, important to emphasize that restriction, constriction, and tamponade may vary in degree of severity and can also coexist, creating clinical pictures which may sometimes be difficult to sort out.

Valvular Function and RV Inflow Tract and RVOT Gradients

The study of RV function would be incomplete without the assessment of the tricuspid or pulmonary valves function. Tricuspid regurgitation may be primary or, more commonly, secondary to RV dilation or pulmonary hypertension. The most common causes of increased right atrioventricular gradient are tricuspid stenosis, tricuspid valvuloplasty, or a prosthetic tricuspid valve. Significant pulmonary regurgitation is usually seen after TOF repair or as a consequence of severe pulmonary hypertension. Increased transpulmonary (outflow tract) gradients may be caused by pulmonary stenosis, structural or dynamic RVOT obstructions or by increased RV cardiac output. Recently, dynamic RVOT obstruction has been described as a possible cause of hemodynamic instability in cardiac surgery (Fig. 15; Video 7; please see video clips available at www.anesthesia-analgesia.org).[62] The guidelines from the ASE for the evaluation of tricuspid and pulmonary regurgitation may be found in a review article published in 2003 by Zoghbi et al.[25]

CONCLUSION

Acute RV failure after cardiac surgery remains a major cause of morbidity and mortality. A comprehensive assessment of RV function may improve risk stratification and lead to early management of RV failure. Echocardiography is becoming a mainstay in the assessment of perioperative RV function. Although RV assessment remains challenging, echocardiography offers useful information on RV size, shape, and function. Future advances in 3D echocardiography may further improve the assessment of complex

congenital defects and lead to better quantification of RV size and function.

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The Right Ventricle in Cardiac Surgery, a Perioperative Perspective: II. Pathophysiology, Clinical Importance, and Management

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The importance of right ventricular (RV) function in cardiovascular disease and cardiac surgery has been recognized for several years. RV dysfunction has been shown to be a significant prognostic factor in cardiac surgery and heart transplantation. In the first article of this review, key features of RV anatomy, physiology, and assessment were presented. In this second part, we review the pathophysiology, clinical importance, and management of RV failure in cardiac surgery.
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Acute right ventricular (RV) failure after cardiac surgery continues to be a significant cause of morbidity and mortality. Acute refractory RV failure occurs in approximately 0.1% of patients following cardiotomy, in 2%–3% of patients following heart transplantation, and in 20%–30% of patients requiring left ventricular (LV) assist device (LVAD) insertion.[1] Acute refractory RV failure is also associated with a high in-hospital mortality rate that may reach 70%–75%.[1–3] In recent years, our understanding of postoperative RV failure has improved, leading to better prevention and management strategies.

In the first part of this review series, the anatomy, physiology, and assessment of the RV were discussed. In the second part, we will discuss the pathophysiology, clinical importance, and management of RV failure in cardiac surgery. We recently reviewed the role of the RV in cardiovascular disease.[4,5] In this article, we will review RV function from the perspective of the surgeon and anesthesiologist caring for the perioperative patient undergoing cardiac surgery.

PATHOPHYSIOLOGY OF RV FAILURE AFTER CARDIAC SURGERY

Postcardiotomy RV failure is often precipitated by an element of ischemia and myocardial depression after cardiopulmonary bypass (CPB).[1] Myocardial dysfunction and pulmonary hypertension (PH) after CPB are usually mild and do not lead to postoperative circulatory failure. However, in vulnerable patients, CPB may contribute to postoperative RV failure.[1,6,7] In patients requiring LVAD support, unloading of the LV alters RV size and shape and may lead to RV failure.[8] In heart transplant recipients, donor heart ischemia and preexisting pulmonary vascular disease increase the risk of postoperative RV failure.[9–11] Other factors which may contribute to postoperative RV failure include 1) suboptimal myocardial protection during surgery, 2) long CPB time, 3) RV myocardial ischemia or infarction caused by coronary embolism or coronary bypass graft occlusion, 4) atrial arrhythmias or loss of atrioventricular synchrony, 5) reperfusion lung injury with secondary PH, 6) postoperative pulmonary embolism, 7) preexisting pulmonary vascular disease, 8) protamine-induced PH, or 9) sepsis-associated myocardial depression (Table 1).

When the RV fails, maintenance of hemodynamic stability depends on LV contraction, especially that of the septum, atrial contraction, atrioventricular synchrony, and RV perfusion. Experimental studies have shown that in models where the RV was replaced with a noncontractile patch, the septum was able to maintain circulatory stability as long as the RV was not dilated.[12–15] Although volume loading may improve RV function, excessive volume loading may contribute to low cardiac output through ventricular interdependence (Fig. 1). RV dilation causes a leftward shift of the ventricular septum, increases pericardial constraint, and modifies LV geometry. As a consequence both LV distensibility and contractility may be decreased resulting in reduced cardiac output.[16]

Acute RV failure may lead to systemic congestion and circulatory failure. Tricuspid regurgitation is usually a prominent feature of acute RV failure and may be the result of RV dilation and PH.[4,16] Hypoxemia is

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Table 1. Common Causes of Right Ventricular Failure in Cardiac Surgery

Mechanism of postoperative RV failure	Specific etiologies
Preexisting RV dysfunction	Preoperative RV dysfunction associated with pulmonary hypertension or congenital, valvular or coronary disease
RV myocardial infarction	Coronary embolism (air, thrombus), thrombotic occlusion, graft dysfunction
Postsurgical myocardial dysfunction	Suboptimal myocardial protection, long cardiopulmonary bypass time
Postoperative pulmonary hypertension	Preexisting pulmonary hypertension Ischemia-reperfusion injury Pulmonary embolism Left ventricular failure Excessive blood transfusions
Dynamic obstruction of the RVOT	Volume depletion, high dose of inotropes
Excessive volume loading of the RV	Excessive transfusions or volume infusion Severe tricuspid regurgitation
Acute unloading of the LV	Following LVAD support
Transplantation	Pulmonary hypertension, prolonged ischemic time, acute rejection, obstruction at the pulmonary artery anastomosis
Pericardial constriction	Postcardiotomy syndrome

LVAD = left ventricular assist device; RV = right ventricular; RVOT = right ventricular outflow tract.

often noted in patients with severe RV failure and may occur as a consequence of increased right to left shunting through a patent foramen ovale or increased ventilation-perfusion mismatches associated with low cardiac output. Alternatively, hypoxemia may be a reflection of the underlying pulmonary disease.

THE IMPORTANCE OF RV FUNCTION IN CARDIAC SURGERY

Over the last three decades, emphasis in cardiology and cardiac surgery has mainly been placed on LV function. Recent data also suggest that RV function may improve risk stratification of patients undergoing surgery for coronary artery disease, valvular heart disease, congenital heart disease (CHD), heart transplantation, or in patients requiring mechanical assist devices and those experiencing postoperative hemodynamic instability (Table 2). In contrast to the evidence that supports LV function in cardiac surgery, most of the evidence that supports the importance of RV function in cardiac surgery is based on retrospective or small prospective studies. Variables of RV function have not yet been included in large-scale risk stratification models. Thus, their inclusion in the Parsonnet score or the Euroscore has not been well established.[17–20] The absence of RV functional parameters in large-scale models may be explained by the more challenging assessment of the RV. However, novel indices of RV function, such as the myocardial performance index (RVMPI) or the tricuspid annular plane systolic excursion, may allow more routine inclusion in risk stratification models.[4]

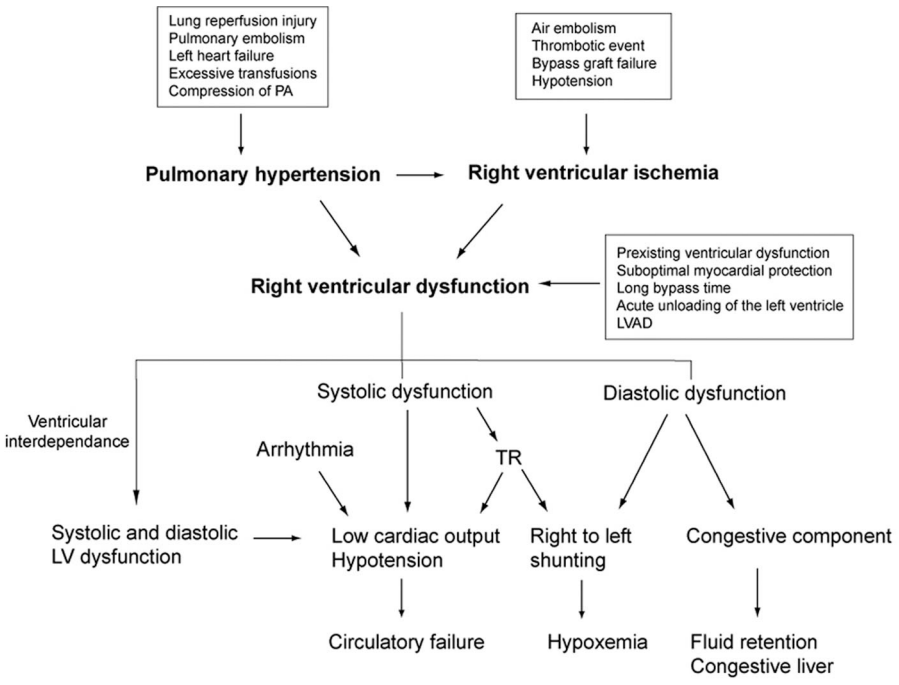

Figure 1. Pathophysiology of right ventricular (RV) failure in cardiac surgery. (LV = left ventricular; LVAD = left ventricular assist device; PA = pulmonary artery; TR = tricuspid regurgitation). Adapted with permission from Haddad F, Denault AY, Couture P, Cartier R, Pellerin M, Levesque S, Lambert J, Tardif JC. Right ventricular myocardial performance index predicts perioperative mortality or circulatory failure in high-risk valvular surgery. J Am Soc Echocardiogr 2007;20:1065–72.

Table 2. Prognostic Value of Right Ventricular Function in Cardiac Surgery

Study	Population	Study design	RV dysfunction	Results
Maslow et al.[21]	CAD undergoing coronary bypass surgery with LVEF <25%	Retrospective $n = 41$	RVFAC < 35%	RV dysfunction is associated with decreased long term survival
Pinzani et al.[22]	Mitral and combined mitro-aortic surgery	Retrospective $n = 382$	Clinical definition	Postoperative RV failure is the strongest predictor of postoperative mortality
Haddad et al.[24]	High-risk valvular surgery	Prospective $n = 50$	RVFAC < 32% or RVMPI > 0.50	Preoperative RV dysfunction was associated with a higher incidence of postoperative circulatory failure
Denault et al.[51]	Patients undergoing cardiac surgery with CPB	Retrospective and prospective $n = 800$	Dynamic obstruction of RVOT (gradient > 25 mm Hg)	Incidence: 4%, dynamic obstruction of RVOT was associated with a higher incidence of difficult weaning from bypass
Reichert et al.[50]	Unstable postoperative patients	Prospective $n = 60$	RVFAC < 35%	RV dysfunction associated with high mortality rates
Webb et al.[34,39]	Atrial septal defect	Retrospective series	RV remodeling	Older age at repair and abnormal RV myocardial relaxation were associated with incomplete RV remodeling
Cullen et al.[42]	Tetralogy of Fallot	Prospective $n = 35$	Restrictive RV physiology	Restrictive physiology predicts longer intensive care unit stay post repair and lower cardiac output
Gatzoulis et al.[43]	Tetralogy of Fallot	Prospective $n = 41$	Restrictive RV physiology	Restrictive physiology predicts smaller RV and better exercise tolerance
Therrien et al.[38]	Tetralogy of Fallot	Prospective $n = 17$	RV remodeling	Severe RV dilatation (RVEDV >170 mL/m^2 or RVESV >85 mL/m^2) associated with incomplete RV remodeling
Kormos et al.[48]	LVAD and RV failure	Retrospective $n = 31$	Clinical mean RVEF = 11.8%	Preoperative clinical factors such as fever, pulmonary edema and intraoperative blood transfusions were associated with RVAD need
Ochiai et al.[2]	LVAD	Retrospective $n = 245$	RV failure requiring RVAD	23 patients (9%) required RVAD. The need for circulatory support, female gender, and non-ischemic etiology predictors of RVAD need.
Hosenpud et al.[9]	Heart transplantation	Retrospective (ISHLT database)	RV failure associated with circulatory failure	RV failure accounts for up to 20% of early deaths

CAD = coronary artery disease; CPB = cardiopulmonary bypass; ISHLT = International Society of Heart Lung Transplantation; LVAD = left ventricular assist device; LVEF = left ventricular ejection fraction; RV = right ventricular; RVAD = right ventricular assist device; RVED = right ventricular end-diastolic volume; RVEF = right ventricular ejection fraction; RVESV = right ventricular end-systolic volume; RVFAC = right ventricular fractional area change; RVMPI = right ventricular myocardial performance index; RVOT = right ventricular outflow tract.

Perioperative Risk Stratification in Coronary Bypass and Valvular Surgery

In patients with ischemic cardiomyopathy and severe LV systolic dysfunction undergoing nonemergent coronary artery bypass surgery, Maslow et al.[21] showed that the presence of significant RV dysfunction was associated with an increased risk of postoperative and long-term morbidity and mortality. Significant RV dysfunction was defined by a RV fractional area change (RVFAC) inferior to 35%.[21] In their retrospective study, patients with RV dysfunction had a higher incidence of postoperative inotropic or mechanical support, required longer intensive care unit and hospital stays, and had reduced short-term and long-term survival rates.[21]

Retrospective or small prospective studies also support the prognostic value of RV function in patients undergoing valvular heart surgery. In a retrospective study of patients undergoing mitral and mitral-aortic valvular surgery, Pinzani et al.[22] demonstrated that preoperative RV failure was a strong predictor of perioperative mortality. In this same study, postoperative RV failure was the most important independent predictor of late survival. In a small prospective study

of 14 patients with severe nonischemic mitral regurgitation and reduced LV and RV function, Wencker et al.[23] found that preoperative RV ejection fraction ≤20% was associated with late postoperative death. In a small prospective study (n = 50), Haddad et al.[24] have shown that preoperative RV dysfunction, as assessed by RVFAC or RVMPI, was associated with postoperative circulatory failure.

The Importance of RV Function in Patients with PH

PH is a major risk factor for mortality in patients undergoing cardiac surgery.[17,25] PH is included in both the Parsonnet and Euroscore models, where it significantly contributes to risk stratification.[17,25] In noncardiac surgery, PH has also been associated with worse outcome.[26,27]

In both the surgical and medical setting, there is growing evidence that morbidity and mortality associated with PH is dependent on RV adaptation to pulmonary vascular disease rather than on the absolute value of pulmonary arterial pressure.[24,26–29] In patients undergoing mitral valve surgery, RV dysfunction defined by a RVMPI >0.50 or a RVFAC <32% was a better predictor of postoperative circulatory failure than pulmonary arterial pressure.[24] In patients undergoing pulmonary embolectomy for chronic thromboembolic PH, studies suggest that preoperative RV failure, manifested by increased right atrial pressure, has been recognized as a risk factor for perioperative mortality.[30,31] Recent studies, however, emphasize that postoperative residual PH is the most important predictor of death.[30] In patients undergoing lung transplantation for PH, reverse cardiac remodeling and recovery of RV function occurs in most of the patients. Predictors of persistence of RV dysfunction have not been yet established.[32] In patients with PH undergoing noncardiac surgery, RV dysfunction, as assessed by the RVMPI, was also a better predictor of survival than absolute pulmonary arterial pressure.[26]

Congenital Heart Disease

The RV plays an important role in CHD, where it may support the pulmonary circulation (pulmonary RV) or the systemic circulation (systemic RV).[4,33,34] Recent studies have demonstrated that RV function is one of the most important predictors of survival and postoperative outcome in patients with CHD and RV pressure or volume overload.[34–37]

In patients with isolated atrial septal defect (ASD) and normal pulmonary pressure or mild PH, closure of the defect usually results in progressive remodeling of the RV.[38] Incomplete RV remodeling in ASD may, however, be seen in older patients (>40 yr old) or in patients with abnormal preoperative RV myocardial relaxation.[38] Atrial arrhythmias in ASD may also persist or develop in adults undergoing cardiac surgery after the age of 40 yr.[34,39] In contrast, closure of an ASD in patients with severe pulmonary vascular disease usually precipitates RV failure. In patients with significant systemic to pulmonary shunting ($Q_p/Q_s > 1.5$) and severe PH, several centers use a preoperative pulmonary vascular resistance (PVR) <15 Wood units and pulmonary to systemic resistance ratio ≤2/3 as a threshold beyond which surgery would carry unacceptable mortality risk.[40,41] However, individual centers vary these thresholds according to pulmonary vascular reactivity and specific anatomic lesions.[41] Closure of the defect is contraindicated in patients with Eisenmenger physiology, unless significant regression of pulmonary vascular disease occurs with pharmacological therapy.

In patients with repaired tetralogy of Fallot (TOF), severe pulmonary regurgitation is the most common cause of progressive RV dilation and failure and is associated with decreased exercise tolerance, atrial and ventricular arrhythmias, and sudden death.[34] Severe RV dilation, especially when progressive, may be the first sign of a failing RV and should prompt consideration of pulmonary valve replacement. Pulmonary valve replacement generally results in a decrease in RV volume.[34] Incomplete RV remodeling with persistence of RV dilation is more common in patients with severely dilated RVs (preoperative RV end-diastolic volume >170 mL/m^2).[38,42] Some patients with TOF exhibit a "restrictive RV physiology," which is defined by the presence of forward and laminar late diastolic pulmonary flow throughout respiration.[34] Early after TOF repair, restrictive RV physiology is associated with a low cardiac output and longer intensive care stay.[34,42] Late after TOF repair, however, restrictive RV physiology counteracts the effects of chronic pulmonary regurgitation. It is associated with a smaller RV, shorter QRS duration, and increased exercise tolerance.[34,43]

Ebstein's anomaly is characterized by an apical displacement of the septal and inferior tricuspid leaflet exceeding 8 mm/m^2.[34] This malformation results in atrialization of a portion of the RV and moderate-to-severe tricuspid regurgitation. The size of the functional RV and tricuspid valve morphology (attachment, commissures, surface) determines the best surgical approach. Preoperative assessment of Ebstein's anomaly is best achieved by combining echocardiography and magnetic resonance imaging.[34,35]

The RV usually adapts well to pulmonary valve stenosis, even when severe. Long-standing severe pulmonary stenosis, however, may lead to RV dilation, RV failure, and tricuspid regurgitation. Percutaneous valvuloplasty is usually considered as the intervention of choice in patients with moderate-to-severe pulmonary valve stenosis.

In transposition of the great arteries (TGA), the anatomic RV supports the systemic circulation.[34,44,45] Because the RV is not well suited to support the systemic circulation, RV failure occurs and is closely related to outcome.[34,44,45] In patients with L-TGA

(congenitally corrected TGA), moderate-to-severe systemic atrioventricular valve (tricuspid valve) regurgitation and RV failure are associated with increased mortality.[34,45] In patients with D-TGA who have undergone an atrial switch operation, myocardial perfusion defects, uncoordinated myocardial contraction, and systemic atrioventricular valve (tricuspid valve) regurgitation may contribute to a progressive decline in RV function.[34,45] Tricuspid valve replacement may slow the progression of RV failure in patients with L-TGA. Late arterial switch operation is also occasionally considered in selected patients, although its benefits have not yet been clearly demonstrated. As patients with TGA get older, many of them may be considered for heart transplantation or mechanical support.

Heart Transplantation

Despite advances in the perioperative management of heart transplantation, acute RV failure still accounts for a significant number of early complications and early deaths in up to 20% of patients in some reports.[9–11] Many factors contribute to the development of acute RV failure and include 1) preexisting or acquired PH, 2) marginal organ preservation and long ischemic time, 3) mechanical obstruction at the level of the pulmonary artery anastomosis, 4) significant donor-recipient mismatch with a much smaller donor heart (more than 20% mismatch in size), and 5) acute allograft rejection.[10] Several studies have demonstrated that preoperative PVR ≥6 Wood units and transpulmonary gradient ≥15 mm Hg are associated with increased perioperative mortality, most probably associated with an increased incidence of acute RV failure.

Left Ventricular Assist Devices

Acute RV failure after LVAD insertion occurs in approximately 10%–30% of patients and is associated with a high mortality.[2,46,47] Many factors may contribute to acute RV failure after LVAD insertion. The most important factor appears to be the acute unloading of the left heart, which results in shifting of the ventricular septum, potentially altering RV geometry, and contractility as well as worsening tricuspid regurgitation. The septal shift results in lower work of the septum and lower septal contribution to RV contraction. This is especially important in patients with baseline RV dysfunction. Predicting acute RV failure after LVAD insertion has proven to be challenging, although several reports have greatly increased our understanding of this problem.[2,46,47] Ochiai et al.[2] reviewed preoperative data for 245 patients, who underwent LVAD implantation. The most significant predictors for RV assist device (RVAD) use after LVAD insertion were the need for circulatory support before LVAD insertion, female gender, and a nonischemic etiology. The most important hemodynamic variables predictive of RVAD use were low-mean pulmonary arterial pressure and low RV stroke work index.[2] Low pulmonary artery pressure in this context was most probably a reflection of a failing RV incapable of generating high, or even normal, pulmonary artery pressures. In a smaller study, Kromos et al.[48] found that preoperative clinical factors, such as fever, pulmonary edema, and intraoperative blood transfusions, were better predictors of RVAD requirements than preimplantation measurement of RV function or hemodynamic variables. A larger study with adequate power to establish a reliable composite predictive risk factor for improving outcome in these patients would be helpful.[47]

Unstable Postoperative Patients

In the hemodynamically unstable postoperative cardiac surgery patient, RV dysfunction is a frequent finding. Costachescu et al.[49] found that RV systolic dysfunction, defined by an RVFAC ≤25% or severe RV dilation, was present in almost half of the patients with hemodynamic compromise. In patients with hypotension requiring inotropic support after cardiac surgery, Reichert et al.[50] have demonstrated that RV failure, defined by RVFAC of <35%, was associated with a high mortality rate. In their study, patients with biventricular failure had a mortality rate as high as 86%. This contrasted with a mortality of 30%–40% for patients with predominately LV failure and a mortality of only 15% for those with normal RV and LV function who were hemodynamically unstable.[50] More recently, dynamic obstruction of the RV outflow tract (RVOT), defined by a RVOT gradient ≥25 mm Hg, was also recognized as a potential cause of difficult weaning from bypass in adults.[51]

PREVENTION AND MANAGEMENT OF POSTOPERATIVE RV FAILURE

Managing acute RV failure after cardiac surgery remains challenging, particularly in certain cardiac surgical settings such as mitral disease, PH, ischemic cardiomyopathy, CHD, heart transplantation, and after the insertion of an LVAD.

Strategies to Minimize the Risk of Postoperative RV Failure

Prevention of acute RV failure after cardiac surgery begins with the identification of high-risk patients. This group includes patients with preexisting PH or preexisting RV dysfunction, patients undergoing surgery with long CPB times, patients receiving cardiac allografts with either long ischemic time or mismatched in size or patients receiving a LVAD.[52] Strategies that may reduce the risk of severe postoperative RV failure include 1) selecting the appropriate timing for surgery, 2) optimizing myocardial protection, 3) selective use of pulmonary vasodilators in the perioperative period, and 4) avoiding liberal transfusion strategies and the use of older blood products.[53]

Avoiding hypotension, optimizing preload, and careful adjustment of ventilation settings by avoiding hypoxemia and hypercapnia are also important principles in the prevention of postoperative RV failure.

Timing of Surgery

Choosing the appropriate timing for surgery may significantly decrease the risk of postoperative RV failure. In patients with valvular heart disease or CHD, cardiac surgery should be considered before the development of severe RV dysfunction develops.[4,54] In patients presenting with an acute RV myocardial infarction, it is usually considered reasonable to delay coronary artery bypass graft surgery for 4 wk to allow for the recovery of RV contractile performance. This approach is suggested if early reperfusion either percutaneously or with thrombolytics cannot be achieved.[55]

Myocardial Protection

Over the last five decades, advances in myocardial protection have significantly improved outcomes in cardiac surgery.[56] Several studies have shown that warm cardioplegia may improve myocardial protection and postoperative outcomes.[56–59] No study, however, has studied the effects of warm cardioplegia on postoperative RV function using echocardiography.

In heart transplantation, continuous perfusion of the donor hearts may reduce ischemic time and allow better myocardial protection and recovery. Continuing prospective studies (PROCEED trial) will determine whether this new strategy will reduce the incidence of postoperative RV failure and improve graft function.

Surgical Approach

Tailoring the surgical procedure in high-risk patients could potentially reduce the incidence of acute RV failure or postoperative RV remodeling. In patients with coronary artery disease and RV dysfunction, the integrity of the RV blood supply must be considered for revascularization.[1] Some surgeons recommend that revascularization of RV marginal arteries may need to be included in the revascularization plan, not only for long-term perfusion but also for delivery of cardioplegia during a cardiac surgical procedure.[1]

Off-pump surgery could have the theoretical benefit of improving myocardial protection in high-risk candidates although an unexpected increase in pulmonary pressures may occur during cardiac manipulation.[60] In low-risk patients undergoing coronary artery bypass surgery, Pegg et al.[60] have recently shown that early and late postoperative RV function was similar between patients undergoing on-pump or off-pump coronary artery bypass surgery.

In patients with functional tricuspid regurgitation associated with dilation of the tricuspid annulus, there is growing evidence that favors repair.[61] Several studies have demonstrated that annuloplasty of the tricuspid valve based on tricuspid dilation improves functional status independent of the degree of tricuspid regurgitation and may improve RV remodeling.[61] On-pump beating heart tricuspid annuloplasty is sometimes considered to improve postsurgical RV remodeling.

Anesthetic Drugs

Studies comparing different anesthetics or techniques on RV function have not been extensively conducted. In clinical practice, various anesthetic regimens have been used successfully in patients with PH who are at risk for postoperative RV dysfunction.[6] In patients with PH, isoflurane is usually recommended for anesthesia, although it may have significant negative inotropic effects when given at clinical concentrations.[62,63]

The Preventive Use of Pulmonary Vasodilators

The prevention of PH and its consequences is a promising strategy for preventing postoperative RV failure. The primary end point in most studies has been pulmonary hemodynamics with limited information available on RV function.

Some investigators believed that the administration of pulmonary vasodilators before CPB could minimize the effects of the pulmonary reperfusion syndrome. In that regard, inhaled prostacyclin and inhaled milrinone have been shown to decrease pulmonary arterial endothelial dysfunction induced by CPB in pig models.[64,65] In small clinical studies, inhaled prostacyclin or inhaled milrinone have been associated with a decrease in postoperative PH. Hache et al.[66] conducted a pilot, randomized, controlled trial in patients with preoperative PH and demonstrated that inhaled prostacyclin given before CPB was superior to placebo in reducing PH. Furthermore, in patients who received inhaled prostacyclin, the amount of vasoactive support was reduced.[67] In a study of high-risk patients by Lamarche et al.,[68] the administration of inhaled milrinone before CPB with PH ($n = 30$) was associated with a lower rate of reinitiation of CPB than when the pulmonary vasodilator was administered after weaning from CPB (9 vs 1; $P = 0.021$). Additionally, postoperative pulmonary artery pressures were lower in the prophylactically treated group. Further prospective and randomized studies will, however, be required to determine the efficacy of this approach. In a recent trial, Fattouch et al.[67] studied patients with PH ($n = 58$) undergoing mitral valve replacement for mitral stenosis. Inhaled prostacyclin and inhaled nitric oxide (iNO) were compared with conventional IV vasodilators. The inhaled drugs were given just before the end of CPB. Inhaled medications were associated with a significant reduction in indices of PH as well as with an increase in cardiac output and in RV ejection fraction compared with conventional treatment. In addition, in both inhaled groups, separation from CPB was easier, the amount of vasoactive drugs administered was smaller, and the duration of stay in the intensive care unit and hospital was shorter. The same

group also compared similar strategies in the treatment of PH after mitral valve replacement upon arrival in the intensive care unit.[67] Inhalation of prostacyclin was associated with a reduction in PVR and an increase in stroke volume. iNO reduced PVR but did not increase stroke volume, and nitroprusside was associated with a reduction in systemic arterial pressure and systemic vascular resistance.

Prevention of Protamine-Induced PH

Inhaled pulmonary vasodilators have also been used to prevent protamine-induced PH and RV failure. In a study of patients undergoing coronary revascularization (n = 3800), Ocal et al.[69] compared two therapeutic approaches in the treatment of the protamine reaction observed in 68 patients (1.8%). One group received inhaled prostacyclin and the other IV nitroglycerin in addition to standard vasoactive drugs. The inhaled prostacyclin group showed improved hemodynamics, and only 14 patients (39%) developing protamine-induced PH had to return to CPB compared with all 30 patients (100%) in the nitroglycerin group. A tendency for shorter length of stay in the intensive care unit and reduced mortality was observed in the inhaled prostacyclin group, but the numbers were too small to be statistically significant. To avoid protamine reactions, heparinase, a heparin degrading enzyme, was compared with placebo in a multicentered, randomized, controlled trial that included 167 patients.[70] The results of the trial, however, were negative. Heparinase was not associated with any reduction in the intervention to treat PH or any reduction in bleeding.

Managing Acute RV Failure After Cardiac Surgery

One of the most important principles in managing postoperative RV failure is to be able to maintain systemic blood pressure while minimizing RV dilation. Maintenance of sinus rhythm and atrioventricular synchrony is also especially important in RV failure, as atrial fibrillation and high-grade atrioventricular block may have profound hemodynamic consequences.[71] Therefore, placement of atrial epicardial leads should be considered in patients at risk of postoperative RV failure. Other important principles include reducing RV afterload, minimizing blood transfusions that may exacerbate PH, and optimizing ventilator settings.[52,72] It is also essential to tailor therapy to the specific etiology of postoperative RV failure (Fig. 2).

Vasopressor and Inotropic Support

In patients with severe postoperative RV failure, vasopressor or inotropic support may be required to maintain hemodynamic stability and prevent the vicious cycle of hypotension and RV ischemia.[1,52] Only a few small studies have compared the efficacy of different vasopressor or inotropic drugs in patients with RV failure or PH after cardiac surgery.

In patients with RV failure and hypotension associated with an acute or chronic increase in pulmonary artery pressure, norepinephrine may be useful in maintaining systemic pressure, increasing cardiac index, and reducing pulmonary pressure.[6,73,74] In an experimental dog model of pulmonary embolism, norepinephrine appeared to be superior to phenylephrine in improving cardiac output.[75] In another experimental animal model, phenylephrine had a negative inotropic effect on RV function.[76] In patients with mild-to-moderate RV dysfunction after cardiac surgery, but without severe hypotension, dobutamine or milrinone are both recommended and may increase cardiac output while decreasing pulmonary pressures.[6,77] In patients with low cardiac output syndrome after cardiac surgery (cardiac output below 2 $L \cdot min^{-1} \cdot m^{-2}$ with pulmonary capillary wedge pressure >10 mm Hg), Feneck et al.[78] compared milrinone with dobutamine in 120 patients. In a subset of patients with PH (defined as PVR >200 $dyn \cdot s \cdot cm^{-5}$ or mean pulmonary artery pressure >25 mm Hg), milrinone had a similar effect to dobutamine on reducing PVR and increasing in cardiac index. The pulmonary capillary wedge pressure and systemic vascular resistance were reduced more significantly by milrinone. Enoximone, an imidazole phosphodiesterase inhibitor, and the combination of dobutamine and nitroglycerine were evaluated in patients with mitral valve regurgitation and pulmonary venous hypertension.[79] Both regimens had comparable effects on mean systemic arterial pressure and heart rate, but enoximone was more effective in reducing mean pulmonary artery pressure. In patients with severe RV failure associated with circulatory failure, epinephrine may also be considered if patients fail to respond to norepinephrine with or without the addition of dobutamine.[80] If a dynamic RVOT obstruction is noted when using large inotropic support, it is usually recommended to reduce the dose, as it may lead to paradoxical decreases in cardiac output.[51] Levosimendan, a calcium sensitizer, has been used in low-output heart failure,[81] but its promise, specifically for RV failure, has been explored only in small human studies[82] and an experimental model.[83–85]

Optimization of RV Preload

In patients with postoperative RV failure and low filling pressures (right atrial pressure <15 mm Hg), preload should be optimized to increase cardiac output. Excessive volume loading should be avoided, however, as it may lead to LV dysfunction through the mechanisms of ventricular interdependence. It may also diminish the contribution of septal contraction to RV function.[1,6]

Inhaled Pulmonary Vasodilators

Inhaled pulmonary vasodilators may be helpful in the treatment of postoperative PH or RV failure. The most commonly used inhaled pulmonary vasodilators

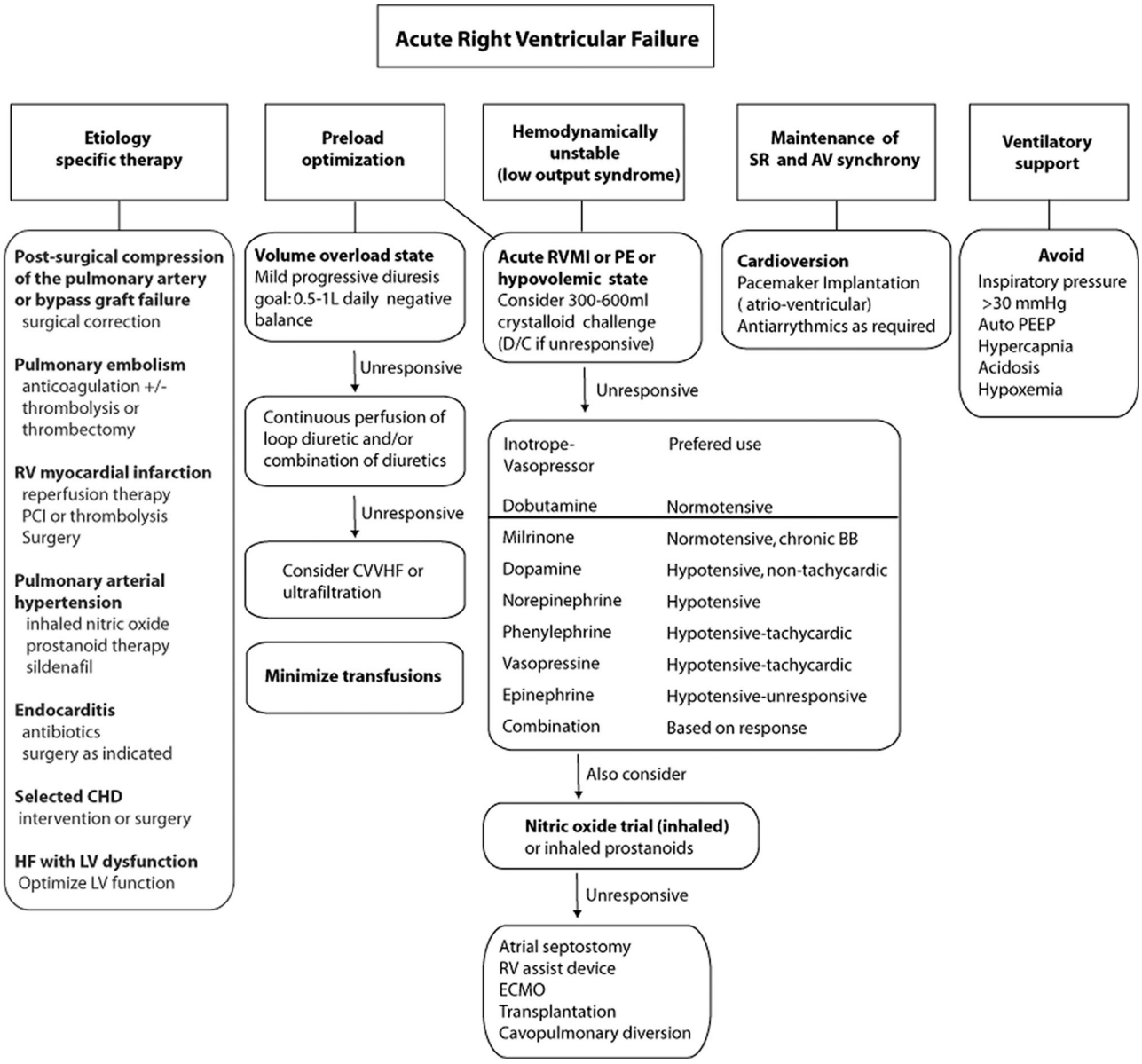

Figure 2. Proposed approach to acute right ventricular failure. AV = atrioventricular; BB = β-blockade; CHD = congenital heart disease; CVVHF = continuous veno-venous hemofiltration; D/C = discontinue; ECMO = extracorporeal membrane oxygenation; HF = heart failure; L = Liter; LV = left ventricular; PCI = percutaneous coronary intervention; PE = pulmonary embolism; PEEP= positive end-expiratory pressure; RV = right ventricular; RVMI = right ventricular myocardial infarction; SR = sinus rhythm. Adapted with permission from Haddad F, Hunt SA, Rosenthal DN, Murphy DJ. Right ventricular function in cardiovascular disease. I. Anatomy, physiology, aging, and functional assessment of the right ventricle. Circulation 2008;117:1436–48.

include iNO, inhaled prostacyclin, iloprost, and inhaled milrinone.[6,86–89] Inhaled sildenafil has been also recently studied in experimental models of lung reperfusion injury.[90] Compared with systemic drugs, inhaled drugs have the advantage of improving pulmonary hemodynamics without increasing ventilation-perfusion mismatches or causing systemic hypotension.

Evidence from retrospective studies and small randomized trials suggest that iNO may decrease the incidence of refractory postoperative RV failure after heart transplantation or CHD surgery.[87,91–94] In patients requiring LVAD support, Argenziano et al.[95] demonstrated in a randomized crossover trial of 11 patients with severe RV failure that iNO led to significant improvement in hemodynamics. RVAD insertion was required in one patient and was partially attributable to abrupt discontinuation of iNO.[95] Retrospective studies also suggest that the use of iNO decreases the need for RVAD insertion after LVAD insertion.[87,96] Comparative studies on different inhaled drugs suggest that iNO and inhaled prostacyclin have equivalent efficacy.[87] Inhaled prostacyclin may have the advantage of lower cost and does not cause methemoglobinemia.[87]

Ventilation

Optimal ventilation settings in patients with severe RV failure may increase RV preload and decrease RV afterload. In general, hyperinflation of the lungs should be avoided, as it may significantly increase

Table 3. Selected Reports of Right Ventricular Assist Device Use for Right Ventricular Failure[a]

Author	Surgical procedure	No. of patients with RVAD use	Survivors
Pae et al.[99]	Postcardiotomy	121	30/121 (25%)
Chen et al.[8]	Postcardiotomy	18/151	6/18 (30%)
McGovern et al.[100]	Postcardiotomy	6/15,000	2/6 (33%)
Pennington et al.[97]	Postcardiotomy	7/4695	0/7 (0%)
Mundth et al.[98]	Postcardiotomy	11	6/11 (54%)
Ochiai et al.[2]	Post LVAD insertion	23/245 (9%)	4/23 (17%) survival to transplant
Barnard et al.[101]	Heart transplant	6	2/6 (33%)
Jacquet et al.[102]	Heart transplant and LVAD	11	6/11 (54%)

[a] Duration of support varied from 2 h to 8 d. Advised caution in patients of age above 70 yr. LVAD = left ventricular assist device; RVAD = right ventricular assist device. Adapted from Kaul TK, Fields BL. Postoperative acute refractory right ventricular failure: incidence, pathogenesis, management and prognosis. Cardiovasc Surg 2000;8:1–9.

PVR.[6] Application of high levels of positive end-expiratory pressure will also narrow the capillaries in the well-ventilated areas and divert flow to less well ventilated or nonventilated areas.[6] Therefore, an increase in ventilation-perfusion mismatch resulting in a decrease in arterial oxygen content can be expected.[6] Hypoxemia, hypercapnia, or acidosis should also be avoided, as they may exacerbate PH.

Surgical Management

In cases where RV failure results from a compression of the pulmonary artery, a stricture at the pulmonary anastomotic site (heart transplantation) or mechanical complication of coronary bypass graft, surgery may correct the underlying cause of failure.[1,52]

Most cases of refractory RV failure will, however, require short-term mechanical support. In the early 1980s, pulmonary artery balloon pumps were used to support the failing RV. Clinical studies have shown that these pumps reduce pulmonary pressures and offload the RV. Because pulmonary artery balloon pumps are less reliable than RVAD, and can only provide short-term support, their use was abandoned in favor of the RVAD.[1] The Thoratec system is the most commonly used RVAD in clinical practice and offers the advantage of longer-term support.[1] Although RVAD reestablishes hemodynamic stability in most cases, the hospital discharge rate following a successful weaning may be as low as 25%–30%[8,97–102] (Table 3). This is in contrast to the 40%–60% salvage rate seen in patients with isolated LVAD support in the acute setting.[1] Occasionally, when time does not allow for surgical insertion of a RVAD or in the presence of associated acute lung injury, an extracorporeal membrane oxygenator is used as a bridge to RVAD or transplantation.[1] In selected cases not considered candidates for RVAD or transplantation and with normal PVR, cavo-pulmonary shunting has been successfully performed in specialized centers.[1]

CONCLUSION

Acute RV failure in cardiac surgery remains a significant cause of morbidity and mortality. Recognition of high-risk patients and early management of RV dysfunction may decrease the incidence of refractory postoperative RV failure. This will be particularly important in high-risk surgeries, such as CHD, multiple valve surgery, and during LVAD insertion. Advances in myocardial protection, prophylactic use of inhaled pulmonary vasodilators, and better RVAD technology will hopefully improve outcomes in this high-risk population.

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